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SAINT BARTHOLOMEW'S HOSPITAL REPORTS.

EDITED BY

DR. ANDREW AND MR. CALLENDER.

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SAINT BARTHOLOMEW'S HOSPITAL REPORTS.

Sir William Lawrence, Bart.

ALTHOUGH the name of Lawrence must ever be amongst the foremost of those which illustrate the annals of surgery, yet there will probably exist much difference of opinion on the exact place which should be assigned to him amongst men who have achieved greatness. For as during life he was remarkable not for the extraordinary development of any one great faculty but for the harmonious combination of various powers which made his character complete, so in future his fame will not rest upon any single brilliant discovery, but upon the record of his whole work.

It is not unlikely that in time to come Lawrence will be ranked by those who never knew him below men who in almost every respect were inferior to him. For in truth it must be confessed that he has left behind him nothing which can convey an adequate conception of what he really was. Even during life his intellectual power and range of knowledge seemed to be fairly appreciated only by the comparatively few who, having the great privilege of working with him, knew him well. Hardly, even now, can the profession estimate the loss it has sustained.

The life of Sir William Lawrence closed on the 5th of July, 1867. He had nearly completed his 84th year. Not more than two years before his death he resigned his active duties at St. Bartholomew's Hospital, but even then he did not

cease from work, and was attacked with apoplexy just previous to an examination at the College of Surgeons.

Lawrence, the son of a surgeon, was born on the 16th of July, 1783, at Cirencester in Gloucestershire. After receiving a preliminary education at a private school, in his seventeenth year he came to London, and was apprenticed to Abernethy, in whose house he resided for five years. But at the expiration of three years, being then only twenty, he was appointed Demonstrator of Anatomy at St. Bartholomew's Hospital. He discharged the duties of this office with remarkable ability for twelve years. When twenty-two, he received the diploma of the Royal College of Surgeons. Eight years afterwards he was appointed Assistant-surgeon to St. Bartholomew's Hospital, and eleven years later still he became one of the principal surgeons. He held this office for more than forty years, and when he retired in 1865, he was unanimously elected by the Governors to the complimentary office of Consulting Surgeon. Thus from first to last he was connected with the Hospital for sixty-eight years.

It is well known that at the early period of his life he worked very hard without interruption, as indeed he did almost to the last. Besides discharging his public duties with rare efficiency, he read very much and wrote too. At eighteen years of age he published an anonymous translation of a Latin work—Description of the arteries of the human body, by Dr. Ad. Murray, Professor at the University of Upsal. In his twenty-sixth year he obtained the Jacksonian Prize for an essay on Hernia. This was the foundation of his important book on the subject, which went through five editions. Lawrence on Ruptures has been for many years past, and will be for many years to come, a standard work on the subject of which it treats. It is remarkable not only for the thoroughly good English in which it is written, and for clearness and justness of thought, but for the mastery of the subject which it exhibits, and for widely extended and accurate research.

In the year he obtained the Jacksonian Prize he published a translation of Blumenbach's Comparative Anatomy. About the same time appeared his paper on Fungus of the Testis. The novel plan of treatment therein proposed was generally accepted and adopted for many years.

In 1814 he was appointed Surgeon to the London Infirmary for Diseases of the Eye, now called the Royal London Ophthalmic Hospital, and in the following year surgeon to the Royal Hospitals of Bethlehem and Bridewell. These offices he held for many years. He devoted a very large share of his attention to the study of diseases of the eye; and in 1833, after many years of patient observation and reflection, he produced his celebrated treatise on the subject, having previously (in 1830) published a smaller volume on syphilitic diseases of the eye. A comprehensive work, written with all the ability and skill which characterised his work on Hernia, it may be said to have marked an epoch in ophthalmic surgery. It was translated into many languages, a portion of it even into Arabic, and went through many editions in English. The last was published in Philadelphia in 1854.

In 1813 he was elected a Fellow of the Royal Society, and subsequently nominated a Vice-President. But it does not appear that he ever contributed any paper.

In 1815 Lawrence was appointed Professor of Anatomy and Surgery to the College of Surgeons. There, during the years 1816, 1817, and 1818, he delivered those famous lectures on Comparative Anatomy, Physiology, Zoology, and the Natural History of Man, which astonished so many, and aroused such animosity, that had their author been a man of less capability, he would, beyond doubt, have been ruined for life.

The question at issue between the author and his assailants is not one which can be discussed within the limits of this notice. But it may be safely said, that were such a work to appear amongst us now, it would evoke no censure beyond that which lies within the bounds of fair scientific or literary criticism. The doctrine (if it can be so called) which then prevailed concerning the nature of Life, and the weakness of which Lawrence exposed with an unsparing hand, has long since become a dogma of the past; and in the discussion of this great question it is only fair to state that he was far in advance of his time. And, again, his view of the other chief subject, the relation of mind to brain, which was then denounced so fiercely, is (whether right or wrong) identical with that of many of the most enlightened physiologists of the present day. In scientific argument, Lawrence was more than a

match for his opponents, but he was assailed by weapons which have been since happily discarded by the champions of knowledge. It may, however, be urged in excuse for the grievous misrepresentation to which he was subjected, that he was not always sufficiently careful to guard against being misunderstood, and proclaimed what he then, and to the last, believed to be the truth, with disregard of, or indifference to, the convictions of those who were then looked up to as the leaders of thought.

In 1816 Lawrence published 'An Introduction to Comparative Anatomy and Physiology, being the two Introductory Lectures delivered at the College of Surgeons on the 21st and 25th of March, 1816;' and in 1819 he published 'Lectures on Physiology, Zoology, and the Natural History of Man.' This last was the celebrated volume Lawrence was subsequently induced to suppress; but, in 1823, Carlile, without the sanction or consent of the author, indeed in spite of anything he could do to restrain him, printed and published a volume entitled 'Lectures on Comparative Anatomy, Physiology, Zoology, and the Natural History of Man,' which included and was simply a copy of the two volumes mentioned above. Many other editions, variously modified, afterwards appeared. The last, called the ninth, was published by Bohn in 1848.

Lawrence taught at the Aldersgate School of Medicine in 1826–27, but he retired from this in 1829, when he succeeded Abernethy as Lecturer on Surgery at St. Bartholomew's Hospital. He occupied this chair for thirty-three years.

In 1828 he was elected on the Council of the College of Surgeons, and in 1840 one of the Examiners, and subsequently he was twice President of the College. Moreover, he became a foreign Associate of the Institute of France, and a member of a host of other societies. Then he was appointed, at first, Surgeon Extraordinary, and afterwards Sergeant-surgeon to the Queen. Finally, he received a title.

Besides the works already mentioned, Lawrence wrote much. He contributed no less than eighteen papers to the Transactions of the Medical and Chirurgical Society, of which he was for many years a member, and, at one time, President. He wrote many of the articles on Natural Science, some of them at a short notice, in 'Rees's Cyclopædia.' He contributed also to

a work of Watt, entitled 'Anatomico-chirurgical Views of the Nose, Mouth, Larynx, and Fauces.'

In 1863 (he was then in his eightieth year) was published his last work, 'Lectures on Surgery.' This, in one volume, by no means embraces the whole course, but only that part of it which was devoted to what may be called the more general subjects, such as the nature of disease, inflammation and its consequences, fever, wounds, and specific diseases. This charming book appeared perhaps somewhat after its time; at all events, it would have produced more effect had its able and accomplished author consented to numerous pressing solicitations to undertake the task—the materials being ready to his hand—many years before. But as it is, better late than never. It is enough to say of it that therein are embodied the most matured experience of the author, and the conclusions at which he had arrived or the opinions he had formed on some of the largest questions which arise in surgery; and moreover it is written with such soundness of judgment and felicity of expression, that it is not only a highly instructive, but a very attractive volume. It is the work not only of a surgeon but of a scholar.

On two instances Lawrence delivered the Hunterian oration at the College of Surgeons. On the last, when the great majority of those whom he addressed were hostile to the views he set forth, his power of public speaking was subjected to a test to which perhaps few men would have proved equal. For the second time in his life did he on a great occasion arouse the anger of his auditory, and excite against himself bitter feelings of resentment. The opinions he expressed on certain questions which were then agitating the minds of many in the profession, and the contempt with which he spoke of certain existing institutions, called forth from all parts of the theatre loud expressions of dissent and denunciation. Probably most of those present thought that the orator would shrink from so unequal an encounter, and bow to the verdict which was so unequivocally pronounced. But Lawrence never for an instant wavered from his purpose. He, standing alone, calm and unshaken by the storm which raged around him, proceeded in a strain of unfaltering eloquence to the end, and concluded with a peroration of such power and beauty that those who had,

during the hour, been loud in condemnation, could not resist joining heartily in the burst of applause which greeted the close. Undoubtedly he then exhibited some of the greatest qualities of an orator.

Lawrence was indeed richly endowed by nature, and he spared no pains to turn his great advantages to good account. Throughout his long life he enjoyed almost uninterrupted health; for within not many months of his death, he declared that he had never been kept from his duties by illness for a week together. His excellent health, notwithstanding hard work, continued through so many years with scarcely any intermission, although no doubt originally the result of an unblemished constitution, was yet, from first to last, carefully preserved by habits of singular regularity and rare power of self-control. No man could be less self-indulgent than he, no one was ever more orderly in his work or punctual to his engagements.

He was, in later life at least, a remarkably handsome man. Above the ordinary height, elegant in form, with a strikingly fine head and face, a magnificent forehead, rising almost vertically above the brow to an extraordinary height, a clear blue eye, which, preserving its brightness even to the last, spoke not only of health and mental vigour, but of a life of uniform temperance, a mouth ample, but with thin lips, and singularly expressive, the distinguished presence of Lawrence as he moved amongst us—a model of intellectual beauty—is not likely to be soon forgotten. He had a natural grace and dignity of bearing, which never failed to impress everyone. Even strangers, when introduced to him, felt at once that they were speaking to a finished gentleman, and before long they found out, further, that this courtly gentleman was a most intelligent and accomplished man. The grandeur of his mental powers, when they were put forth, threw all his other gifts into the shade. His was a vast and capacious intellect, and singularly complete. It seemed equal to the grasp of any subject brought before it, and it had been so long and sedulously cultivated that no subject seemed to come strange to it. There was a rare charm in his conversation. He touched nothing that he did not adorn. Whether it were philosophy or history, science or literature, there was still the same powerful and penetrating

thought, the same unfaltering fluency of pure and perspicuous language. But he was the least egotistical of men. He would talk on any subject but himself, and mingled with all he thought and said there was the additional attraction of a quiet, half-suppressed wit and humour, oftentimes too subtle to be caught by all; not seldom a refined and polished sarcasm, which, usually oblique and playful, was occasionally, it must be confessed, when he was bored by a fool or provoked by a dunce, terribly bitter and direct. Many, particularly those who fell under it, thought there was too much of this. He was, certainly, very often careless of whom he in this way offended; and there is no doubt that most of his enemies could trace back their resentment to some few words of his which they could not forget, and would not forgive. Thus there were not a few who would pour their vials of wrath upon Lawrence—behind his back; and the indisposition which he always evinced to speak of himself or of his acts rather encouraged such to gossip and scandal. Since his death, some of these have taken a bolder flight, and have not hesitated to stoop to gross misrepresentation, and to vent their spleen by publishing the basest calumnies. Were Lawrence still living to hear of these, they would probably affect him hardly more than they disturb him now.

Dr. Joseph Bullar, in an admirable memoir of Lawrence, says: 'In a notice of Sir Wm. Lawrence's life in the "Times" an old "canard" was revived, that he had, after withdrawing his Lectures on Man from circulation, sold them for sale in America. This story having been shown to be entirely untrue, the writer of the notice then asked, "Why have the statements, which have been so long before the public, remained unchallenged until now?"

'The reason is that Lawrence made it a rule not to contradict such reports, as I well recollect.

'When I was his dresser in 1829–30 he was accused of taking a fee from an out-patient. Mr. James Farish, his house surgeon, who was present, knew that a well-to-do city patient, to suit his own convenience, had called on Mr. Lawrence at St. Bartholomew's Hospital, and he asked Lawrence to allow him to explain the circumstance. "No," said he, "I have made it a rule never to answer such attacks."'

Dr. Bullar adds: 'Lawrence was a strong man with a clear-headed consciousness of his predominating ability, and such men are apt to treat such accusations with silence. Controversy (says Oliver Holmes) equalizes fools and wise men—and the fools know it.'

Perhaps many who imagine that they knew Lawrence very well never understood him. One of the noblest traits of his character was his almost sublime indifference to effect. There was something fascinating in the power of mind and range of knowledge which he, on almost all occasions, seemed to hold in reserve. Never was there suspicion of what is commonly called 'claptrap' about Lawrence. He was not mere surface, with every quality and attainment ostentatiously laid out, like trinkets in a shop front, to catch the public eye. If you sought, and he would permit you, to penetrate his stores of knowledge, you found more costly gems of thought within, but these he never cared to display.

To the last he was true to the habit of his whole life. To the last he not only read much, and remembered to an extraordinary degree what he read, but thoroughly digested it, separating with remarkable skill the wheat from the chaff. But although he must have thought very much, he wrote, for such a man, comparatively little, and hence our loss in him is all the greater, and irreparable. Only a few weeks before he resigned his post at the Hospital, a turn in conversation led him to speak of the modern systems of philosophy, and of the most recent works on the subject. It soon became evident that he had not only diligently read and thoroughly mastered them, but that he had also reflected deeply on their doctrines and tendencies.

But as his habitual reticence and reserve caused him, during his life, to be but poorly appreciated even by many of those who knew him best, so the works he has left behind him, distinguished as they are and always will be for the knowledge and mastery of the subject they exhibit, for vigour of thought and clearness of exposition — his published works, notwithstanding these excellences, but faintly represent the mind which produced them. It is admitted by those most competent to judge that he wrote well, that his style was excellent—correct, perspicuous, and graceful. But, again, it will be acknowledged

that he was greater as a lecturer than as a writer, and those who had the best opportunity of judging will probably agree with Sir Benjamin Brodie that he was greater in ordinary conversation than in public speaking. Many pages might be filled with illustrations of his power of argument and of his wit and humour, subordinate though these were to his higher qualities. Unquestionably he sent forth materials for a much better volume of table-talk than any which has yet appeared. But Lawrence was not merely occasionally good in conversation. He was not like celebrated talkers generally—at times brilliant, but too often feeble and commonplace. He was remarkable, not so much for rare flashes of great power, producing startling effects, as for the uniform excellence of his talk. Surely no man ever said so few weak things or was so seldom silly as Lawrence. If he, bowing to the common infirmity of our nature, was not wise at all hours, in these barren intervals he had at least the discretion to be silent. There was nothing of the 'prating fool'—of the tale-bearer—about him. But he could not be fairly charged with want of candour. He was not false by withholding the truth. If Lawrence was secret, he was not insincere.

In him the passions and emotions, which so disturb and excite ordinary mortals, had but little sway; they seemed ever in complete subjection to his will. He was never the victim of uncontrollable impulse. All his acts, even in emergency, were the result of deliberate calculation, of calm and unruffled reason. Whatever errors Lawrence committed in life were probably errors of pure judgment. It could not be said that in him reason was ever obscured or overruled by passion. This absence at all times, even when most tried, of any visible emotion, made him appear cold; while his habitual reserve repelled all confidence. Hence, while he compelled men to admire him at a distance, he did not attract them closely or win their affection. Yet, if Lawrence did not habitually exhibit a warm heart, he was rigidly just and upright in his dealings, and those who were best acquainted with him and his acts know that when he appeared to be harsh or ungenerous, it was from a clear and firm conviction, whether that were right or wrong, that he was acting fairly and justly. Such conduct may not, indeed, always have seemed amiable, but it was almost

always true. Lawrence hated and despised all cant and imposture, and easily saw through them. He possessed singular skill in estimating the ability and attainments of others, and was not readily imposed on in this or, indeed, in any way. But while he contemptuously trampled ignorance and assumption under foot, he was quick to appreciate, and would willingly advance intelligence and industry.

'He could be very generous,' writes Dr. Bullar, who knew him intimately. 'He gave his favourite nephew (whose life of high promise as a surgeon was cut short by the cholera of 1831) unlimited leave to draw on his banker for expenses at the medical schools of France and Germany; and an Irish gentleman, who apprenticed himself to Lawrence at a later age than usual, told me that Mr. Lawrence strongly dissuaded him from spending his fee (500*l.*) in this way.'

It is a matter of course that a man like Lawrence would attain extraordinary eminence in whatever profession he worked, and to say that he diligently devoted his life to the practice of surgery and to the sciences upon which it is based, is simply to affirm that he became one of the greatest surgeons that have ever ennobled it. His was no low or narrow view of the requirements of surgery; its practice in his hands was no mere empirical or mechanical art, but he thought it worthy of the highest powers of the intellect. He was no mere skilled artisan, but a thorough philosopher. Nor was he content to cultivate the sciences only so far as they were directly applicable to practice; but he loved them for their own sake, and from first to last he pursued them for the charms they possessed. The zeal with which he studied, especially in early life, anatomy and physiology, human and comparative, is well known. He never failed, throughout his long career, to possess himself of the current knowledge of these sciences, and, to the last, it was most interesting and instructive to hear him discuss the doctrines of the day.

One afternoon, during the last year of his tenure of office at the Hospital, he was met by an old pupil in the theatre of the College of Surgeons, where he was attentively examining some ancient skulls, which had recently been placed there for exhibition. The few remarks he then made were sufficient to show how well qualified he was to have spoken publicly upon

some of the difficult questions which at present engage the attention of physiologists.

As a surgeon Lawrence was distinguished rather by his clear and vigorous conception of principles than by any special or peculiar skill in the details of practice. This is well illustrated in his last work—the volume of Surgical Lectures which he published. His great ability was never more conspicuous than in prognosis. At times, with marvellous dexterity, he would predict the issue of a case which baffled or misled every one around him. Thus his most daring achievements as an operator were often crowned with success, which to others appeared in the distance as the remotest chance.

His practice was remarkable for decision and effect. His treatment of a case was very seldom 'expectant.' When he acted it was always to some purpose. He prescribed very few drugs, and only those of unequivocal power. Yet he was very far from being a rash surgeon, and no man was ever less meddlesome than he. Usually very cautious and far-sighted, his mistakes in operative surgery were comparatively very rare; but—and this was characteristic of the man—when they did occur, were sometimes of considerable magnitude. His superiority was most apparent in consultation. Usually he said very little, but that was always strictly to the point. At such times he rarely indulged in speculation, but addressed himself only to the practical questions which arose. His opinion and advice were eminently reasonable. Even when you differed from his conclusions you felt the force and soundness of the views he took. And then many may remember how, by a few trenchant remarks, he would scatter to the winds an elaborate hypothesis, or expose the fallacy of a diagnosis by a single question. It is significant that those who knew him were restrained by his presence and became unusually slow and cautious in advancing an opinion.

As an operator, before his physical powers began to fail, he was decidedly skilful; but his skill was shown, not so much in grace of action or in apparent manual dexterity, as in the ability with which he accomplished the end in view, and the manner in which he brought the operation to a close. Near-sighted in early life, his vision in his declining years, except towards the end, not only did not fail, but in some respects

improved. To the last he operated without glasses, and those, which for some years he occasionally used at other times, still later he almost entirely laid aside.

All his colleagues and pupils, and many others, were familiar with his amazing coolness and perfect self-possession under the greatest difficulties. He seemed never to be disturbed, even by the most perplexing and untoward occurrence. Beyond the faintest blush, which, perhaps, now and then for a moment passed over his countenance, no indication of any kind appeared that he was in the least degree affected. This wonderful equanimity made him the same at all times, and under every circumstance. Whatever he may have occasionally felt was known only to himself. So far as observation went the worst difficulties of an operation or the storm of an indignant assembly alike failed to evoke any evidence of emotion.

In the way he did his work at the Hospital Lawrence taught a great lesson. He spared himself no pains or trouble. Whatever might be the stress of his other engagements, no sign of it appeared in the orderly, regular, and punctual manner in which he performed his public duties. Even when busiest he always found time, without apparent hurry, to do well and thoroughly his hospital work. And then his bearing to the sick poor was absolutely faultless. Always kind and considerate, no one was ever more gracious and gentle to women and children. It was delightful to witness his tenderness to them, and it is pleasant to recall it. But no matter what the condition or antecedents of any patient might have been, Lawrence never inflicted unnecessary pain even by a rough word.

In speaking of Lawrence as he worked and taught at the Hospital, I gladly accept the opportunity which the kindness of Dr. Latham has offered of introducing some remarks on him as a clinical teacher, which were written many—perhaps twenty—years ago by one of the very best of all his pupils. It is indeed fitting that what William Ormerod thought of Lawrence should be recorded here. He says:—

'The few years spent by a student at a large hospital afford great opportunities of collecting definite practical knowledge of individual cases of diseases, and of testing the applicability of the more important principles of treatment. Still the years are few, the attention is divided amongst a number of objects,

whilst the necessity of being prepared for approaching examinations renders the time devoted exclusively to the acquirement of clinical knowledge short, interrupted, and too often insufficient. How immensely valuable, then, is the teacher who by example and precept is constantly setting before the student those principles of observation, which may to a certain degree supply the shortness of time, and indicate the only true path of useful and proper study, which may in fact render a partially educated man capable of gradually educating himself during the daily duties of the practice of his profession. This Mr. Lawrence did in an eminent degree. The whole tenour of his writings, conversation, and practice was to set before the student the value of the great practical writers in the profession, and the utter worthlessness of the mere noisy claimants for notoriety, to teach all to form a proper appreciation of the constant and hard labour required for a proper and competent discharge of professional duties, and the real incapacity of anybody not practically laborious, and above all to lay clearly before every one that whilst professional learning allowed us to deal with some of the greatest difficulties and dangers, that there was a clear line bounding our knowledge, beyond which we had no right to assume that our information extended. To be ignorant of clearly ascertained and written knowledge, to be idle or not absolutely industrious, or to attempt to hide ignorance by assumed learning, or a mere guess by obstinate certainty and noisy argument, were with him faults of so deep a dye, and so utterly below all honourable feeling, that his countenance at once told the student his hatred and contempt of such vile attempts at assuming professional character and knowledge. If a few years or months only were allowed the student to attend the practice of Mr. Lawrence, in that short time he could learn that he had entered a hard and laborious profession, where every faculty should find its use in searching the recorded experience of the present and past generation, in studying disease by treating it, and in rendering himself competent to detect the indications of treatment in difficulties, to seize the characteristic distinctions in questions of obscure diagnosis, and to meet the dangers and emergencies of great operations. To reach this position the student could learn from his example that he was able and should daily persevere, but

the practice and advice of his accomplished and accurate teacher taught him that to order active means without indications, to be wise without reasons, and to operate for mere operating's sake, were but the signs of rashness, inexperience, and folly. How often have we waited at the ward fire for Mr. Lawrence in his week, though not on his day, feeling sure that his love for his work and daily diligence had not forsaken him, and that he would come to see if anything was wanted. Perhaps some injury of the head, a bad fracture, or a case of acute inflammation had been admitted, some case which had a real difficulty and required an immediate decision, or perhaps even an operation. As he entered the ward each hat was taken off, and the sister standing at the right-hand of the door curtseyed, but nobody spoke. Mr. Lawrence then bowed or nodded to somebody, or to the house surgeon, and then proceeded to the bedside of the patient, to whom a few kind words were spoken before anything was done. An examination of the patient was then gone through, with a few questions to the house surgeon, to the sister, or to the patient himself, and then the orders were given, or the patient was told that some operation was required, or nothing was said; but the short question, "Is Mr. So-and-So here?" told the regular attendants that a consultation would be held. But this short interview at the bedside was often succeeded, as we knew, by a few remarks at the fireplace away from the beside, where a short clinical lecture on the case and its probable termination, with especial stress on the points to be watched as capable of being treated, and a minute description of any rarity either in the occurrence of such cases or in any particular symptom present were, as we well knew, so often given and so good, that we calculated on them.

'How often have we rejoiced to see him in the theatre, whether he operated or not. If he operated himself everything was done with the greatest care, from the time at which he tried the edge of the scalpel on his thumb to the moment at which the patient left the room, unencumbered with any quantity of bandage and plasters and other ill-timed applications. If he did not operate himself, we knew how sure we were to have a good view, for nobody so carefully cleared the area between the patient and the class of opaque intruders, and we knew that when he was by, persons were very shy of say-

ing anything without being quite sure of it. These were our gain as pupils, but his colleagues found in him a ready assistant, and such an adviser on the spur of the moment as is rarely met with, for the learning and quotations from Italian, German, and French authors, which fill his written works, were never permitted to overlie or interfere with the constantly ready and eminently practical knowledge which a life spent in the wards of this country's greatest hospital had so amply given.

'The surgeon of a large hospital holds a most responsible position. The relief of the sick committed to his charge is his first duty, the education of pupils and the advancement of professional knowledge are also required of him; but for the relief of his patients and the instruction of those intrusted to him he is bound to give the best and highest degree of professional knowledge. The day's duty which raises a poor man from the bed of sickness may supply precedents for the pupils and be the means of multiplied blessings to others in future years, as surely as their omission may allow not one patient only, but ultimately hundreds, to linger on uncured or even perish. The successful lecturer and the learned or practical author are, for these reasons, far inferior in usefulness to him, who, knowing his profession, practises it publicly, and daily transacts before the eyes of a large class the great experiments of the healing art. The rule may admit of some exceptions, as in the case of one great living medical writer, and in such rare minds as Harvey, Jenner, and Hunter; but the influence for good of Pott, Abernethy, and Sir Astley Cooper, who wrote well and largely, and of Cline, Mr. Blizard, and Mr. Liston, who wrote but little, is probably greatest and widest by the daily instruction which a steady and laborious attendance to their hospital duties afforded to a large and studious class of pupils. Such was Mr. Lawrence's great usefulness, though he lectured, and wrote, and spoke, each in a manner equalled by few, and in all together probably surpassed by none. The student who diligently attended his practice saw a man of the highest abilities, of knowledge, of minute accuracy, and never-failing readiness, punctually attending to his duties in a large hospital, to the patients of which he afforded that care and relief which laborious education and long experience, with cool-

ness in the boldest and delicacy in the most minute operations, could alone convey. The bookworm or the practical man, the lover of the operating theatre or the mere dexterous manipulator, might all learn a lesson from him, when a rare dislocation afforded scope for reference to authorities, a cancer of the breast allowed him to quote his long and decided experience, a severe case of erysipelas, or some formidable tumour of doubtful relations, formed the subject of his operations, or some delicate operation for hernia or cataract attested that delicacy of touch which his great accuracy in the recognition of deep-seated matter so often showed him to possess.

'Was Mr. Lawrence a student through life? During the earlier years of his life, and during the writing of his books and the arrangement of his lectures, he undoubtedly was, and a most laborious one. The character of his writings indicates deep and close study; the printed surgical lectures are a good summary of the best practical authors, and his own knowledge of professional literature was great. Still this complete knowledge, with certain exceptions, did not seem to extend beyond a certain date, and those numerous and voluminous octavos which for so many years have been inundating the medical public were but slightly known to him, though he seemed to know the main points so well, that even the working student was not too willing to be questioned by him in conversation on them. He seemed by hard study and constant clinical observation to have laid after some years a most solid and extensive foundation of surgical knowledge, unencumbered by doubts, resting on the most certain proofs, quite sufficient for practice, and far beyond all ordinary men; this he daily practised and perfected more and more, from it he was willing to remove what was shown by clear evidence to be wrong, and to it he seemed to be adding from time to time by selection and well-chosen reading the principal practical points of recent date.

'The character of each man's mind retains through life the impress of the tone of the period in which he was educated, and the beginning of this century was indeed one of importance in our profession. The works of Hunter were probably more studied then than now; the human body, though not so generally known by the microscope, was perhaps more accurately by the eye; and anatomy was studied in relation to the great

questions of surgical operations. The connection of clinical medicine with surgical practice, the relation of fever, of tubercle, of organic disease of the internal organs, and of the various important morbid diatheses as evidenced by the urine, with the ordinary cases of disease treated by surgeons, were only then beginning to be generally studied; whilst the mass of minute medical chemistry and microscopic pathology, which during the last few years has almost daily, and often almost too rapidly, been laid before the profession, was all but utterly unknown. But, without all this, it was a great and important period, as great indeed as the present. To it we owe the mass of our knowledge of the anatomy of hernia and of fractures and dislocations; from it we have gained our best information on the diseases of the eye and the injuries of the head; and the really valuable additions to our knowledge on diseases of the skin, the treatment of aneurism, malignant diseases, and syphilis, since that period, though great, have neither crushed nor overbalanced the foundations then laid. If the rising generation are daily showing the wonderful connection between the animal and vegetable kingdom, unravelling by chemistry and by the microscope minutest structures and changes, and thus explaining the highest principles and the most abstruse relations of the secret changes and processes of the human body in health and disease, still their fathers did as great a work when they laid such a foundation as we possess in their works on such subjects as the diseases of the brain, the bloodvessels, and the eye, the anatomy of hernia and of the injuries to the skeleton, and the practical pathology of syphilis and malignant disease.

'It was during this period that Mr. Lawrence was educated, and to these subjects his mind evidently turned. His clinical lectures and his works were the best proof. The volume on the treatment of hernia is the best complete work on the subject in our language, from its excellent account of the anatomy of the parts in health and the important and various changes in disease, based not only on his own dissections and the rich collections of St. Bartholomew's museum, but containing also the purely anatomical researches of Cloquet, Langenbeck, and Camper, with the equally important and well-weighed observations of such anatomists and surgeons as Scarpa, Sir Astley Cooper, and Hey. The work on diseases of the eye

stands less alone amongst the works on this subject than that on hernia; it is equal to any in its accurate descriptions of disease; but, whilst the work on hernia is that of a laborious student and a decided and active surgeon, the treatise on diseases of the eye seems often to be rather the work of a pathologist and accurate observer of disease.'

The intellect of Lawrence was characterised by a rarely equable and harmonious adjustment of parts. There appeared to be no deficiency or excess. All the faculties of his mind, like his frame, were in such perfect proportion, that you were never struck by anything in particular, but charmed with the whole effect. Just as when one looks on the most perfect trophies of art, no part especially catches and arrests the eye, after the fashion of inferior works, but by equality of excellence they at once suppress criticism and command admiration: so with Lawrence. There was nothing monstrous or eccentric about him. In a word, it might be said of him that the whole man, mind and body, was in perfect taste; and—as it is only with the greatest—that the better he was understood the more he was admired.

It is well for some men that they are publicly known only by their works, for it has too often happened that the charm which an author has raised has been rudely dispelled by personal acquaintance. But of men of the stamp of Lawrence no adequate conception can be formed from their books. Lawrence was so much greater in himself than in what he has written, that it can never be said of him, as he said of Hunter, he 'lives again in the ample stores of knowledge left behind him.'

But his name and memory and the tradition of his work he has left—a rich legacy—to the profession to which he devoted his life, and lived so eminently to adorn. His example will survive and influence surgery long after those have passed away who can remember the fine old gentleman as he went round the wards of St. Bartholomew's. And, as the great surgeon of the Hospital, one may borrow for him his own noble image of Hunter, and say, 'Here he reigns alone, like the Jupiter of the heathen mythology—

'Cui nullum viget simile aut secundum.'

Article I.—*Removal of an enormous pendulous Tumour from the Back.* By Luther Holden.

ELIJAH FARMER, æt. 67, was admitted into Colston Ward on July 31, 1867. He is of middle stature, rather corpulent, has always enjoyed excellent health, and has regularly worked at his trade as a plumber and glazier up to the day before his admission.

The object of his coming was to be relieved of an enormous tumour growing from the back between the shoulder-blades, as represented in the cut. Its longitudinal circumference measured 41 in., its transverse circumference 40 in., the circumference of its root 30 in. Its weight was guessed at 35 lbs. The skin over it was natural in appearance. Several arteries as large as the radial at the wrist could be felt pulsating over it, and numerous large and varicose veins were visible upon it.

The tumour had existed for twenty-four years. When first noticed, it was about the size of a walnut. For many years it grew very slowly; latterly it had been increasing in size at a great rate.

As the tumour was probably composed chiefly of fat; as it was tolerably loose and moveable at its root; as the patient was in excellent health and spirits, and extremely desirous to be rid of his gigantic burden, my colleagues with myself came to the conclusion that its removal was justifiable.

The operation was performed under chloroform on August 3. Considering the great bulk and weight of the tumour, arrangements were made to support it by large hooks attached to a rope passed over a pulley. Thus it could not tear itself away during the operation, and one could the more easily work at its root.

Its detachment was accomplished without any unforeseen difficulty. The hæmorrhage from the numerous vessels was arrested by little spring pincers, ligatures being applied after the removal of the mass. The wound was dressed with a compress of dry lint and a roller.

The shock of the operation was not more severe than might have been anticipated. He soon began to rally under the careful watching of his dresser, Mr. F. E. Aldrich, who sat up with him during the first night.

On the third day he had quite recovered from the shock. The wound was dressed for the first time with a lotion of carbolic acid, in the proportion of one drachm of the acid to a pint

of water, which answered admirably throughout the subsequent progress of the case in removing all offensive odour.

On the eighth day the bowels acted for the first time, after an ordinary injection.

Sept. 5.—Since the date of the last note the patient has progressed uninterruptedly well, and the wound is cicatrising and contracting rapidly.

Nov. 6.—Patient left the Hospital perfectly well.

The tumour weighed 37 lbs. It consisted of masses of fat, of various consistence, intersected by strong fibrous partitions.

In the centre of it was a large irregular cavity, containing about two quarts of thick oil, with small lumps of degenerated fat float-

ing in it. Its appearance on a perpendicular section is shown in the annexed woodcut. Two coloured drawings of it, one before, the other after its removal, are preserved in the museum of St. Bartholomew's Hospital.

ARTICLE II.—*On Spasm or excessive Contraction of the muscular coat of the Urethra, commonly called Spasmodic Stricture.* By WILLIAM S. SAVORY, F.R.S.

WHEN stricture of the urethra is spoken of, anyone is understood to mean an abnormal contraction of some portion of the canal from structural change, for although surgeons classify strictures into organic, spasmodic, inflammatory, and so forth, the latter forms hold but a very subordinate place in the literature of the subject, while in practice it often happens that they obtain little or no consideration whatever. Some surgeons, indeed, deny or ignore the existence of spasm altogether; even by our authors of most repute spasmodic stricture is stated to be very rare, and it is hardly admitted that spasm may occur alone.

Not very long ago it was believed that spasmodic stricture, when it occurred, was necessarily limited to the membranous portion of the urethra, where it is encircled by striated muscle, because it was supposed that it could be caused only by the action of the compressor urethræ muscle. Even now the membranous part of the urethra is still spoken of as the muscular portion. Since, however, it has been known that the urethra, in its entire course, is invested by muscular fibre, it has been admitted, though for the most part grudgingly, that spasm may occur in any portion of the canal.

But although it is now allowed that the canal of the urethra may become abnormally contracted by muscular action in any part of its course, the opinion seems everywhere to prevail that the so-called compressor urethræ muscle is the chief agent in the production of spasmodic stricture, and that it is therefore usually limited to and always most intense at the membranous portion of the urethra.

But is this view borne out by observation or analogy?

The idea that the membranous portion of the urethra is the chief seat of spasm rests on the old belief that the part in question alone is muscular, and on the fact that instruments are more frequently obstructed in their passage there than elsewhere.

But then here the canal becomes smaller; there is the fibrous septum called the triangular ligament, and just at or about this part organic stricture is most common.

Evidence of variation of calibre from muscular contraction appears to me to be most decided and best marked in the portion of the urethra anterior to this, not only from variation in the size and shape of the stream of urine in health, but also from the effects often experienced in the passage of instruments. They are sometimes grasped and held with force by the urethra.

No portion of Hunter's surgical works seems to me more instructive than his remarks on this subject. From clinical observation he predicated that the urethra must be muscular throughout, long before the fact was demonstrated by microscopical research.

Assuming spasm to exist in the membranous portion, is it then due to the contraction of the compressor urethræ muscle? Are not the unstriped fibres still concerned in its production?

We do not attribute certain forms of colic or spasmodic asthma or excessive contraction of the smaller arteries under certain circumstances, to any other agent than the unstriped muscular fibre in the walls of the tubes, because this is the only form of muscular tissue present. Is not, then, the muscular coat of the urethra equally competent to produce all the effects of spasm which are witnessed?

Is not the character of the contraction better explained by the action of the plain than of the striated muscle? Does not the comparatively abiding or equably sustained character of the spasm rather accord with the action of the plain fibre? Is not spasmodic stricture of the urethra more akin to the inordinate contraction of other tubes which can only be acted on by plain fibre than to the intermittent or oscillating character of the cramp with which striated muscle is often affected?

With reference to the assertion that examples of pure spasmodic stricture are very rare, of course in every case there must be something wrong to produce excessive or spasmodic contraction, and it is the business of the surgeon to discover and correct the mischief. But if by pure spasmodic stricture is meant the occurrence of spasm apart from inflammation or structural contraction, it cannot be conceded that the statement is accurate. I believe there is evidence to show that not only is inordinate contraction of the muscular coat of the urethra commonly, nay, almost constantly, associated with stricture from structural change, but that undue muscular contraction of the urethra apart from the presence of any structural change—or

simple spasmodic stricture—is much more common than it is supposed to be, its effects being often attributed to permanent stricture.

That spasm is commonly associated with permanent stricture is shown by—

The varying size of the stream even when permanently diminished. How very few instances of stricture occur in which, upon investigation, it does not appear that the stream of urine varies much in size; that at certain times and under favourable circumstances the urine is passed much more easily and quickly than at others. This fluctuation in the size and other characters of the stream which is so very common in cases of stricture cannot be due to any permanent alteration of the canal, but must be referred to some temporary cause which is capable of modifying its size and shape. In this way may be recognised, I think, the fact that abnormal contraction or spasm of the muscular tissue of the urethra is commonly associated with or engrafted on permanent contraction of its canal; nor does it appear difficult to understand how such extraordinary contraction of the muscular coat should be provoked by the additional force with which the urine is necessarily driven into the urethra against the stricture—

The greater facility with which instruments may be introduced at some times and under certain circumstances than at others—

The speedy improvement which follows measures that can hardly, in so short a time, affect a permanent stricture; such as rest, warmth, aperients, opium—

The effect of removing the pressure of the urine from behind, as by tapping the bladder. This is often at once followed by partial relaxation of the contracted portion of the canal. Beyond the immediate relief which is afforded by the operation this appears to be the great advantage which is derived from puncturing the bladder or urethra behind in retention of urine from stricture. The urine is no longer impelled against the obstruction by straining, and the muscle of the urethra, allowed to rest, relaxes—*

The effects of enlarged prostate. Sir B. Brodie remarks: 'Where a simple chronic enlargement of the prostate gland supervenes on stricture of the urethra, the latter usually becomes less liable to spasm, and is more easily dilated, and altogether more tractable than it was before.' Because now

* See especially Cock, 'Medico-Chirurgical Transactions,' vol. xxxv., and 'Guy's Hospital Reports,' 3rd series, vol. xii.

the pressure of the urine, which was a constant source of irritation, is intercepted by the prostate—*

The fact of complete retention of urine suddenly supervening upon stricture—†

The fact, often verified after death, that the degree of organic contraction will not account for the degree of obstruction which exists.

Thus, I think, can be explained how the controversy about impermeable stricture has arisen. Much discussion has been expended upon the question whether such a condition as a really impermeable stricture ever occurs. That the canal of the urethra is ever so permanently closed and sealed up by organic stricture as to be altogether impervious to urine may very well be denied; but no position can be less tenable than that taken by those who argue that where urine can pass out an instrument may be passed in. In some cases, indeed, neither urine nor instrument can pass; but even if the former can somewhat freely escape it by no means follows that the latter can be passed through. This is a very different thing, for setting aside other considerations everything may depend upon the presence or absence of muscular contraction. There may be a passage for the urine, but it may be closed against an instrument.

With reference to the fact of spasm or inordinate contraction supervening upon organic or permanent stricture, the question suggests itself—Is not muscular contraction least capable of affecting the calibre of the urethra where the walls of the canal are most indurated? It would appear that in proportion to the rigidity of the walls so must the effect of spasm be diminished.

There can be no difficulty, however, in understanding that structural change of the wall of the urethra producing permanent stricture, whether associated or not with congestion or inflammation, may be the means of exciting undue or spasmodic contraction of the muscular coat of adjacent or distant parts of the urethra.

But, further: I do not think that considerable narrowing of the canal of the urethra from contraction of its muscular tissue alone is very rare. Where there is no difficulty in voiding the urine such diminution of the stream is apt to escape observation, but cases not unfrequently occur in which the fact cannot be overlooked. For example: I lately saw a man of middle

* Works, 1865, vol. ii. p. 404.

† See Brodie, op. cit., 1865, vol. ii. pp. 395 and 413.

age who has been in the habit for many months past of introducing at intervals of ten days or a fortnight a catheter into his bladder to prevent, as he expressed it, the canal closing. He assured me that he had more than once attempted to give up the practice, and the difficulty and distress of voiding the urine by a slender stream became gradually so great that he was always at length driven to seek artificial relief. This man had no evidence whatever of organic stricture or of any permanent obstruction, but I have seen the stream vary in size from that of a No. 10 or 12 to that of a No. 2 or 3 catheter. It may be mentioned that other means, such as the warm bath and opium, always afforded him temporary relief, but he preferred the use of the catheter. I do not mean to justify, nor did I sanction, the use of any instrument in this case which very likely assisted to provoke the mischief it was intended to remedy, but I think there can be little doubt to what the remarkable diminution of the stream was due.

Again, I lately saw a younger man whose stream of urine varied even more than in the last case. Here the stream was rarely even moderately full, and often so scanty that it was only by a great effort that it became continuous. The water would often in great part dribble away. Yet a large instrument might be always, even at the worst times, introduced without any difficulty into the bladder, but it was for awhile firmly grasped by the whole urethra. I could mention other cases of the same kind.

On the other hand, cases might be cited where the urine flowed freely in a full stream, and yet where the passage of an instrument was obstructed. A man was in the Hospital complaining of symptoms which led to the use of the catheter. The urine flowed in a full stream, and if a full-sized instrument were quickly and lightly passed it went smoothly into the bladder, but when withdrawn it was impossible to repass it for some time afterwards. Its previous passage had induced so much irritability that it was at once firmly grasped. If not adroitly introduced this would often occur in the first instance. It might indeed be suggested that owing to clumsy manipulation the extremity became lodged against some point of the wall of the urethra; but it was obvious to all who saw the case that the passage of the instrument was not thus obstructed but that it was held tightly by the urethra.

That spasm therefore may and often does occur alone is shown by the frequent and very considerable diminution of the stream of urine—which, however, often escapes notice—and by

circumstances which sometimes attend the passage of an instrument.

Spasm when admitted is regarded as a very transitory affair. Hence the contrast of the term permanent, which is given to stricture due to structural change.

That spasm or muscular contraction of any degree is in its very nature transitory there can be no doubt; but yet cases occur in which all the symptoms of stricture endure for years, but in which, nevertheless, there is clear evidence to show that it entirely depends on muscular contraction.

In the following case the spasm very closely simulated organic stricture.

A man between thirty and forty years of age, whose health for some time previously had been unsatisfactory, complained of considerable difficulty in passing urine. Very much straining was needed to relieve the bladder, and then the urine only in part escaped in a very slender stream and the rest dribbled away. The gentlest introduction of any instrument always gave acute pain, and the point was arrested at a variable distance, but usually within some four or five inches of the orifice. So sensitive was the urethra that the canal could not be fairly examined without chloroform, and even then, on almost every occasion, the instrument was arrested before it reached the bladder. Only once or twice was this accomplished in the earlier part of the case, and then an instrument of moderate size —No. 7 or 8—was carried through with facility. This state of things endured for many months, and it was observed that the condition of the urethra always varied with the general health. Now and then, to the man's astonishment, the urine would pass freely in a full stream, but in spite of every apparent precaution the amendment was only transitory; in a few hours the difficulty and distress were as great as ever. After a time all attempts to pass an instrument were given up except when some fresh surgeon was consulted, who of course did not believe in the difficulty until he himself had made an unsuccessful attempt to overcome it. Then, some time after the urethra had been left to itself, the symptoms gradually abated. The first indication of improvement was that during a loose action of the bowels, after aperients, the urine would escape in a full stream without difficulty. At times a tolerably free stream would flow for days together, and then again, perhaps from some indiscretion in diet, the condition of parts would become as distressing as ever. Even to the last any attempt to introduce an instrument was always unsuccessful and mischievous. But

at length the evil gradually abated, and for the last year or two the urine has been passed freely in a full stream.

In the 'Lancet' of April 13, 1867, Mr. Campbell De Morgan has recorded a case of what may well be called permanent spasmodic stricture inasmuch as it was of some twenty years' duration.

The explanation of these and of many other cases more or less persistent and unyielding, I take to be this: The muscular coat of the urethra cannot always be in a state of spasmodic or inordinate action, but is immediately excited to contract unduly by any impression upon the mucous membrane of the urethra or adjacent parts: so the contact of urine, or still more of an instrument, at once provokes it. Thus, as the canal is almost always more or less contracted by the passage of urine, and still more completely closed by any attempt to pass an instrument, evidence of stricture always exists when its condition can be examined, and thus, so far as every appearance is concerned, the contraction is persistent, but in all probability there is in intervals of complete quietude more or less complete relaxation.

The influence of chloroform is sometimes very great in these cases, and its value twofold—in diagnosis and in treatment. It is not always, however, sufficient to cause thorough relaxation of these muscular fibres.

When spasm is associated with organic contraction or inflammation or congestion, either alone, or still more when combined, it may be reasonably assumed that the cause of it is obvious enough; but if spasmodic stricture may and often does occur independently of any of these, the question arises—To what cause may spasm, thus pure and simple, be due?

The exciting cause of simple spasm may be either in the urethra itself, or it may arise in neighbouring or even in distant parts. A common example of the former class is the spasm which is often provoked by the introduction of an instrument. Everyone knows how the effect is increased by clumsy manipulation, and how the difficulty is often averted by attention to the temperature of the instrument, and, better still, by gentleness and lightness of hand. As before mentioned, the difficulty may be often escaped, too, by rapidly carrying the instrument into the bladder, taking the urethra, if one may so speak, unaware. So, again, the distress which frequently follows the employment of irritating injections, or the passage of irritating urine, as sometimes after the application of a blister, are examples of the same kind. As illustrations of the

latter class may be mentioned the spasm associated with inflammation of the prostate, and that which is often induced by some affections of, or operations on, the rectum, or even by fracture of the leg. The degree to which these several exciting causes may act will of course depend upon the disposition of the urethra itself.

It is significant that spasm of the urethra is not uncommonly associated with spasmodic action in other parts. At present I have under observation an elderly man, in whom attacks of spasm of the urethra, leading to retention, alternate with attacks of spasmodic asthma.

If there is truth in what has been advanced, it follows that the treatment of stricture of the urethra is very often too mechanical, and consequently apt to lead to much mischief. No doubt in the investigation of any case the distinction between organic and spasmodic stricture is usually borne in mind, although, indeed, as already mentioned, there are some who ignore the existence of spasm altogether; but when the conclusion has been arrived at that there is a permanent contraction of the canal, perhaps too little care is given to ascertain whether there is simply a thickened, indurated, and contracted state of the mucous membrane, the result of morbid action which has now entirely passed away, or whether there is not still some evidence of inflammation or congestion or spasm. Not only is there a difference here between precept and practice, but even in the literature of the subject a strange inconsistency often appears. While in the pathology of stricture spasm is acknowledged to be a frequent complication of permanent contraction, in the consideration of treatment its almost constant co-existence is practically ignored. The management of organic stricture is discussed as if there were no such condition as spasm.

Is sufficient attention given to the pain which is often caused even by the most careful and gentle introduction of an instrument? Would an organic stricture in a passive state explain such tenderness of a particular portion of the urethra? Are we, at all times, cautious enough to avoid making the urethra bleed? Whenever blood follows the introduction of an instrument, is it not a sign that, in one respect at least, mischievous force has been employed?

If this be true, should not all attempts to cure permanent stricture by mechanical means be delayed, until, by previous measures of a very different kind, all active mischief in the form of inflammation or congestion has entirely passed away;

until even all spasm has been, so far as it is practicable, subdued; and until no morbid condition beyond simple passive stricture—contraction due solely to structural change—can be detected? No doubt this will be assented to in theory, and by some considered trite, but is it carried out as it should be in practice? May I be allowed to say that I do not presume to address these remarks to those who, admitting the soundness of the principle, invariably act upon it.

In point of fact, I scarcely ever meet with a case of stricture which is in a fit condition for immediate treatment by mechanical dilatation of any kind. After the nature of the case has been ascertained, it seems to me almost invariably to require, before any attempt whatever at mechanical dilatation is made, a period of absolute rest with or without other measures according to circumstances. And however highly we may estimate the value of rest and recumbency in these cases, I think we must be continually struck by the great benefit derived from it. A person who previously can only void his urine in drops by most severe and painful efforts, and is in instant danger of complete retention, after rest for a few days will pass urine in a stream with comparative comfort.

If I may venture to speak in general terms, I would say that, in the treatment of cases of stricture, the rule should be to try first what can be done by other measures before resorting to mechanical means. The introduction of an instrument may be indeed necessary in the first instance as a means of diagnosis, often enabling us moreover, by observing its effect, to determine whether it shall be employed, and if so when, in the further treatment of the case.

It need hardly be remarked that in the treatment of spasmodic stricture when not associated with any morbid change in the part—when pure and simple—the cause of it, whether direct or indirect, must be sought for, and if possible removed.

Beyond the all-important means of rest and recumbency and warmth in the form of baths or otherwise there are, we know, remedies which have powerful action in directly subduing spasm—chloroform for instance, which in cases of emergency will sometimes signally succeed, and enable us without difficulty to pass a catheter through an urethra which just before had proved impermeable. Opium, which in these cases is so often an invaluable remedy, as a rule perhaps is best administered as laudanum by the rectum; but this or some other form of it, whether solid or fluid, may be given by the mouth, or morphia may be injected under the skin. Belladonna,

too, or its alkaloid atropine, is sometimes useful, especially when locally applied; but, as a rule, I think they bear no comparison in efficacy to opium.

Active aperients, too, are often strikingly serviceable. The urine, which up to the period of their action had been obstinately retained, will sometimes pass freely during a sharp action of the bowels. The escape of urine, which is often simultaneous with the action of the bowels, and usually then most free, is a fact which should be always borne in mind. And it may be remarked that the success which attends the use of such remedies as those which have been indicated is an additional proof, and a strong one, of the nature of the case. But, let it be repeated, we should, whenever it is practicable, go farther than this in our treatment of spasm of the urethra, and discover if possible the cause on which its occurrence depends.

Even when spasm is associated with structural change or organic stricture, it may be controlled by the same means, more or less actively employed according to circumstances, although of course their effects are not now so striking or apparently satisfactory. When there is evidence of further mischief in the form of inflammation or congestion, local depletion or perhaps counter-irritation by other means than the application of cantharides may be advisable. But except in extreme and very urgent cases the use of drugs and of all active remedies may well be dispensed with, and reliance placed on simple measures of hygiene, such as wholesome food, warmth, and, above all, rest for a while even in bed.

In conclusion, it is only right to state that the substance of this paper was written more than five years ago, and that further experience during this period has confirmed the opinions I have ventured to express. However faulty these opinions may be, they are therefore hardly open to the charge of hasty publication.

Article III.—*On the Pathology of Fatty Degeneration.*

By Edward Latham Ormerod, M.D. Cantab.

Fatty accumulation and fatty degeneration, the physiological process and its pathological counterpart, are not to be placed in strong contrast, or regarded as two functions alto-

together distinct and practically separable from each other. On the contrary, dissimilar as they may be in some of their phases, they shade into each other at so many points that no exact line of demarcation can be drawn between them. They are directed by the same chemical principles, and the results, as measured by the same anatomical rules, are essentially identical.

Not to lengthen these remarks by an enquiry into the laws of fatty accumulation, it is at least necessary, here, at the outset, briefly to indicate the successive steps by which the physiological merges into the pathological process.

Regarding the subject from a chemical point of view:—

1. In a condition of health, where all the organs are as yet sound and the years few, only such alimentary substances as are not available for immediate use are turned into fat. Obesity in this condition generally results from an excessive use of non-nitrogenous food. The physiological problem here is precisely the same as that which a smoky lamp presents, the carbon in both cases being supplied out of due proportion to the oxygen, and being deposited in the one case as fat, in the other as soot. And a properly devised regimen and diet are as surely successful in preventing the formation of fat in the animal, as adjusting the burner and the feeder are in preventing the formation of soot by the lamp.

2. It is a step lower than this when fatty accumulation results from a deficiency of oxygen rather than from an excess of carbon, when the fat is supplied by the carbon which enters into the composition of the nitrogenous as well as of the non-nitrogenous elements of the food. The remedies here are not so obvious nor so easily applicable, for the system may not be able to bear any considerable diminution of the class of aliments which supply the fatty matter in this case. We cannot leave these off as safely as we can suspend the use of non-nitrogenous articles of diet; and, in advancing years, with which this second stage may probably coincide, the organs have lost something of their original integrity and reparative powers, and answer less readily to remedies.

3. The third stage into which this insensibly shades is lower still. Here the source of the fat is removed yet farther from our control; for it is not formed from the food introduced from without, but is a product of the daily waste of the tissues. This is the lowest condition of suboxidation. The invading substance itself is more widely diffused, and permeates other parts besides the cells of the common adipose tissue. Our remedial measures are now utterly inadequate to meet the

difficulty. The principles to be applied are indeed the same, but their practical application is often quite impossible.*

Again, regarding the subject from an anatomical point of view, following the guidance of Virchow,† we find that fatty matter may accumulate under three typical forms:—

1. In the first of these, which common adipose tissue familiarly illustrates, the cells in which the fatty matter is deposited are adapted for its permanent reception. This is a normal condition, the pathological equivalent of which occurs in the form of fatty tumours, and of local fatty accumulations which resist the common physiological influences to which adipose tissue generally is subordinated. The accumulation, except, usually, in the case of fatty tumours, may arise, and be dissipated, and arise again, without any detriment to the integrity of the containing tissue, whose function is to separate and retain fat for the purposes of the system.

2. The second typical form under which fat accumulates is as a temporary infiltration of parts to whose functions it is incidental to be thus temporarily loaded with fat or oil, which is permeating them for transmission onwards. Such parts are the epithelial glands of the intestines and of the liver. When in active operation they are loaded with fatty matter, and when they have discharged their share of the digestive function the fatty matter passes on and they are free. But when, from any cause, this transmission is delayed, and the oil is retained within the gland cells, and the fatty infiltration becomes permanent, then the physiological line is passed, and a morbid condition results, which is most familiarly illustrated by fatty degeneration of the liver.

3. The third form of fatty accumulation is where this substance is infiltrated into parts which do not contain it in any appreciable quantity in the physiological condition, and where the infiltration is accompanied by the disorganisation of the part. The typical normal illustration of this form is supplied by milk, where epithelium cells are produced in great abundance, only to be infiltrated with fatty matter, and then to be thrown off and die, producing milk as the result of this disorganisation. This form of fatty accumulation is most fully illustrated in pathology. Instance the fatty infiltration of cells which results in the formation of the compound granular cell. Such, again, are the changes in muscular fibre and in the renal

* See, on all this subject, Dr. H. Bence Jones's 'Lectures on the Applications of Chemistry, &c., to Pathology and Therapeutics,' 8vo. 1867, p. 170.

† 'Cellular Pathologie,' S. 288.

epithelium, which will be considered in detail in the further progress of these remarks.

From both these points of view, anatomical and chemical, fatty degeneration seems to stand as one of the last of a series of retrograde changes by which health shades into disease. Anatomically traced, the gland-cells which should transmit fatty matter under these circumstances retain it, till that which should be a store of available nourishment becomes a permanent mechanical burden, and a hindrance to the functions of the part affected. Again, chemically traced, we find that in fatty degeneration the materials of the food and the products of the waste of the tissues are diverted from their usual destination. Their attraction for oxygen is limited by their chemical affinities, rather than measured, as heretofore, by the physiological requirements of the system generally. They are suboxidised, in chemical language, and take the form of fat.

As we descend in the series, from health to disease, the physiological force, which in the first instance rose superior to everything, and made itself felt throughout, attracting to the growing germ all that was required for its perfect development, sensibly fails. Its life-long antagonist, chemical affinity, now asserts its superiority in turn, so that fatty degeneration, in which this growing superiority seems at last to culminate, has even been regarded by some as a purely chemical change independent of all vital influences. I would venture to make a few observations on this, as it seems to me, untenable hypothesis.

First, generally. We may often find substances of fatty origin belonging to the stearic or oleic series, as, for instance, acetic, lactic, or oxalic acid, or the rancid secretions in acute rheumatism or other febrile affections, occurring as the products of disease in the living body, the results of a degradation of the higher members of their respective series. But we cannot imitate Nature's operations directly in the laboratory. We can neither ascend the scale and make fat from the lower members of the series, nor can we suspend the process of degradation at any given point; nor yet can we change a member of one series into another at will. That is quite beyond our power as chemists. The very limited success which we do reach in such experiments is obtained by working, so to say, with Nature's own tools—by putrefaction, for instance, rather than by a less complex operation. And, practically, the results are as yet rather curious tricks of the perfumer's trade than ava lable applications of scientific chemistry.

It could hardly be otherwise. The powers by which organic

substances are formed, converted, and decomposed in the ordinary physiological processes are wholly unlike those which we bring to bear upon them in our chemical experiments; and the results of Nature's operations are wholly unlike ours. To go no further than the instance before us, the strong affinity by which the elements of the hydrocarbons are bound together, their permanence and their resistance to anything short of destructive chemical agencies, contrast strongly with their extreme plasticity under the influences of the living body. By gentle continuous physiological influences results are obtained which violence and direct chemical action cannot procure. There is the same difference between Nature's chemistry and our own as between leading and driving, as between the gentle breaking up of the rocks by frost and the splitting them by gunpowder. Nature does most by the vis a fronte; our operations are mainly applications of the vis a tergo.

When living materials are acted on, one set of changes takes place, which chemistry can interpret but cannot imitate. But when the physiological influence is withdrawn after death, and chemical affinity has full play, we have another set of products quite different to those which appear during life. Taking our illustration, again, from the subject before us, no known chemical process could make wax out of the honey or sugar in the stomach of the honey-bee. The heat indeed, the moisture, the insufficient supply of air in the dark hive while the swarm is making wax, are all more or less direct chemical agencies which tend to the conversion of the honey into wax. But these must act in live bees to produce the result. The whole process ceases with the death of the bee. Chemical affinity is not the ruling power while life lasts, it only becomes so when life has ceased. Then, under its influence the elements of the body take new forms and move in new directions, the bees fall from their curtain, they undergo putrefaction, and the honey in their stomachs is converted, not into wax, but into alcohol and carbonic acid.

Again, more particularly: observations of certain changes in dead animal matter both without and within the living body have been adduced in favour of this opinion. I. The conversion of dead animal substances by gradual decomposition into a matter known by the name of adipocere has been cited as an instance of the conversion of animal substances not containing fat into fatty matter. II. And the accumulation of fatty matter in and about certain extraneous substances introduced into the cavity of the peritonæum of living animals has been

accepted as a proof that the same conversion may take place within the living organism. I think that neither of these conclusions will stand the test of examination.

I. With regard to the adipocerous transformation. Adipocere is a brittle waxy-looking substance, differing in its chemical characters according to the source from whence it has been obtained. This is the substance so familiar to us in our Museums, the eye-sore of anatomists. We may find it, whenever we want some, in the cavities of the long bones, or in the nervous foramina of the skull. The easiest way of making it is by exposing a large mass of animal matter to the action of very slowly running water. There are other methods more available on a small scale, all acting on the same principle of decomposition at a low temperature, under water, with limited access of air; but adipocere, as familiarly known, is generally obtained by this particular means.

A stock of adipocere for examination was procured from the different sources which the macerating jars of the Sussex County Hospital Museum supplied, and from a large specimen in the Museum of the Royal College of Surgeons. For the liberality with which my wishes were met, and a portion of this specimen placed in my hands for analysis, I beg to express my sincere thanks to the Committee of the College Museum, and to their zealous Curator, Mr. Flower, whose kindness on this, as on other occasions, I have much pleasure in acknowledging.

1. Adipocere obtained from the neighbourhood of the bones of the head of a sword-fish was found to be composed of margaric and oleic acids; the latter in excess, combined with lime. There was a small quantity of unaltered fat and of free fatty acids, also some undecomposed nitrogenous matter.

2. From adipocere which had gathered round human bones, the same general results were obtained; only the margarates preponderated over the oleates, and the saponification was more complete. This last result seemed due to the longer time during which the specimen had been macerating. The adipocere obtained from these sources and from a mass of human muscle burned with a clear flame, leaving a white alkaline ash; but the fat from fatty hearts burned entirely away without any residue.

3. A portion of adipocere from the human thigh (prep. No. 1832*d*, Mus. Coll. Surgeons) was submitted to a most careful analysis, to test in some way the accuracy of the previous observations. It was composed of white stringy fibres powdered

over with a dry substance, which lay partly between the bundles of fibres, and partly in distinct masses. This substance had no definite form, and only obstructed the view of the fibres to which it adhered. When it was removed by ether, or, more completely, by cold liquor potassæ, regular fibrous tissue, just such as is seen in muscular fasciæ, came into view. A very little heat sufficed to gelatinise this tissue.

The nature of the different constituents having been determined on separate portions, an exact quantitative analysis was made of ten grains of the specimen, giving:

Free margaric and oleic acids	4·1
Margarate and oleate of lime, with traces of magnesia . .	2·4
A peculiar fibre, containing nitrogen and traces of phosphate of lime	3·4
Water	0·1
	10·0

In the fatty matter here present, margaric acid so predominated, that it was necessary to add olive oil to allow it to crystallise freely in its characteristic form. In this, as in all other respects, this adipocere agreed perfectly with other specimens of adipocere from the human subject, and contrasted strongly with the results of fatty degeneration.*

Briefly to recapitulate. When adipocere occurs in large masses, as in a whole limb which has been submitted to the action of slowly-running water, and where we have the product of the decomposition of various tissues, we find that there has not been a general uniform change of them all. The least destructible of the various tissues—such, for instance, as the fibrous sheaths and fasciæ—are still to be recognised by their microscopic characters. The more perishable, such as the muscular tissue, are represented, after a while, only by a dry amorphous powder, in which the presence of nitrogen may be detected by the usual tests.

But these remains of different tissues are not adipocere. This is a substance which varies indeed in its composition within certain limits, and which has the special characters of the fat whence it was derived impressed upon it: if, for instance, it has been derived from fish, it consists mainly of oleate of lime;

* The experiments here referred to were made for me by Mr. James Peel, at that time Dispenser to the Sussex County Hospital. I regret much that I have no longer the advantage of his skill in pursuing this enquiry. The details are reproduced from the Medical Address delivered at Cambridge in 1864. 'Brit. Med. Journal,' 1864, vol. ii. p. 154. To this and to some further observations on the same subject ('Brit. Med. Journal,' 1865, vol. i. p. 475) I shall have again occasion to refer.

if from the human subject, margarate of lime is in excess. But, otherwise, it is of an uniform and definite composition. It is, in fact, a compound of the particular fatty acid with an alkaline base, perhaps ammonia, or more commonly lime derived from the water used in the process of conversion. If we employ distilled water in our experiments, and withold an alkaline base, no adipocere will be made, but the fat will remain as fat, and will not be saponified.

Further than this. I believe that no fat whatever is made during the adipocerous transformation. Such was the invariable result of all my experiments made, with one exception, on the muscular fibre of the heart. So far from the fatty matter having increased, it was rather diminished in quantity during the process.

For this purpose, however, we must avail ourselves of some of the other processes for the production of adipocere. For however well adapted the process by slowly-running water be for the production of adipocere on a large scale, it is quite inapplicable where great accuracy is required. When the meshes of the net containing the weighed fragments of muscle were fine they became clogged, and prevented the free access of water, and then the muscle putrefied; while coarser meshes allowed fragments of the softened tissue to escape. Notwithstanding all my care, none of the experiments by this method with weighed portions of muscle, of which the fatty constituents had been previously determined by analysis, succeeded. And after varying the apparatus several times, to no purpose, I gave up this mode of proceeding, and made use of the processes by dilute nitric acid or dilute alcohol, with much more satisfactory results, as follows:—

A portion of the muscular substance of a heart which had previously been found to contain 2·14 per cent. of fatty matter, chiefly margarine, was left to soak for twenty months in dilute nitric acid, sp. gr. 1·42 (1 to 16) in a stoppered bottle. At the end of this time the fatty matters were found to amount to no more than 1·1 per cent. of the five hundred grains operated on. The mass had been changed into a substance having the smell and appearance of ointment of nitrate of mercury. The elements of the fat originally existing in the mass had been newly arranged under the influence of the nitric acid, part having assumed the form of an aromatic ether. Much of the fat had been destroyed; but never at any time during the process was there any evidence of the fat having been increased in quantity.

The muscular fibres generally retained their form, clearly recognisable under the microscope. The fibrils were broken off short into minute fragments; but each of these displayed distinctly either the transverse striæ, or that longitudinal dotting which characterises the stage of granular degeneration already spoken of.

Again, two hundred and fifty grains of pure fibrine from bullock's blood, freed from all fatty matter by ether, were treated in the same way and for the same period. Not a trace of fat, of any of the oily acids, or of the results of the decomposition, could be found on analysis.

A portion of the muscular substance of a heart, which had been previously determined to contain 2 per cent. of fatty matter, was left to soak for eight months in dilute alcohol (1 to 7) in a stoppered bottle. As with the dilute nitric acid, this bottle was nearly filled with the solution, occasionally shaken, but rarely, if ever, opened; and all observations were made on a similar trial solution. At the end of this time the mass was found to contain 1·92 per cent. of fat and fatty salts of whatever kind. The fat seemed to have been destroyed, to this extent, by the process to which the muscular fibre had been submitted.

To the naked eye, the muscle thus acted on appeared as a reddish, flocculent mass, in which the division of the fibres into bundles was indistinctly perceived. Under the microscope, however, all was obscure. A few granular fibrils could be picked out here and there; but the rest was an indistinct, flocculent mass, with something of a linear arrangement. It contained a few white, opaque grains with a radiating structure, apparently composed of a soap of lime. No loose fat appeared anywhere, and ether had very little effect in clearing the field.

I would pass over the effect of slight decomposition on muscular fibre in inducing seeming fatty degeneration, with the remark that, complete as the deception is to the eye, the truth is plain at once on chemical analysis of the suspected structure. In no sense of the word, nor at any step of the process, is adipocerous transformation a conversion into fat.

II. Again: it has been asserted, on the faith of certain experiments, that dead or extraneous animal substances may be converted into fat within the living body. In the absence of the original records of these observations, I quote from Lehmann,* and from a summary by Mr. Simon.† I am quite

* 'Physiologische Chemie,' 2te Auflage, S. 345.

† Holmes's 'System of Surgery,' vol. i. p. 11, article Inflammation.

sure that everyone conversant with the difficulties of experiments on living animals, and sharing my dislike to witnessing them, will excuse my not having repeated them.

The experiments may be divided into three classes:—

1. Portions of animal substances containing little or no fat, as for instance of the crystalline lens, of coagulated albumen, and such like, were inserted into the peritonæal cavities of living animals. After periods of from four to eight weeks they were removed and examined, when they were found much reduced in size and altered in chemical composition, containing very little protein-substance, but a large quantity of fatty matter (Wagner, Michaelis).

2. Porous substances, such as cancellous bone or elder-pith, inserted in the same way, were found, after a certain time, to contain much fatty matter in their pores, and the coagulable lymph with which they had become invested was also rich in fatty matter (Burdach).

3. The same class of substances as were used bare in the first series of experiments, when covered with collodion, or enclosed in glass bottles, underwent no fatty transformation (Burdach).

These experiments seem to me to prove that if the extraneous substance be protected from contact with the fluids of the living body, no fat is deposited in its interior. But wherever this contact is permitted, fatty deposition takes place, and this whatever the nature of the extraneous body. The substance, animal or vegetable, sponge, elder-pith, pumice-stone, or muscular fibre, is infiltrated with fatty matter simply according to its permeability to the fluids of the living body which contain the fatty matter. And there is no more reason to suppose the fatty matty to have been derived from one than from another class of extraneous substances, from the muscular fibre than from the elder-pith. In all of them alike it is merely an infiltration and deposition, deeper and more abundant according to their porosity, and to the facility with which the living fluids bringing fatty matter from the blood can enter them.

The process of fatty degeneration, if I am correct in these inferences, is not adipocerous transformation, nor does it ensue in dead animal substances—dead as we commonly understand the word. What it is may be more positively stated, after we have carefully examined the mode of its occurrence in some of its well-marked forms.

A complete history of fatty degeneration should be coextensive with physiological histology. It would require an account

of how each and every tissue is affected by this morbid process, and a description of their characters severally, under this condition. Instead of following out the subject in such detail, I propose to myself to investigate the general principle which is common to all these manifestations, rather than to recount the varied forms which fatty degeneration may present in different structures. For this purpose it will be necessary to examine thoroughly the process of fatty degeneration as displayed in a few typical instances. And it seems best, on all accounts, to select these instances from the structures which have been made the subject of special study by other observers. In no other way could an equal security against errors, either of observation or of judgment, be obtained.

I propose, in this view, to commence with the instance to which I have paid much attention during many years, namely, fatty degeneration of the muscular structure of the heart, connecting with this the fibre of voluntary muscle. Fatty degeneration of the liver and kidneys supplies two well marked and well studied instances of this process in glandular structures. Fatty degeneration of the placenta offers another instance well adapted to our present purpose, both by the peculiar structure of the organ, and by the fact of this change having been closely examined by other observers. To these instances, having long waited in vain for specimens on which to repeat Mr. Canton's observations on arcus senilis, I have added an abstract of his conclusions without any re-examination.

I trust, at some future time, should opportunities allow, to publish the results of the study of fatty degeneration of some other structures. The pathological conditions known as acute yellow atrophy of the brain, rickets, and cirrhosis of the lungs are those which chiefly invite attention as offering the means of testing the correctness of my present conclusions from different points of view. But the appearance of this paper has been so long delayed from various causes, that I prefer to publish it in its present form, rather than to wait for time to carry out another series of observations. The accordance of all the results hitherto obtained, whether from my own observations, or the independent researches of others, gives me confidence that a farther extension of the enquiry will only tend still more to confirm them.

Fatty Degeneration of striated Muscle.

Fatty degeneration of voluntary or transversely striated muscular fibre has a particular claim to our notice as the instance in which this pathological process has been chiefly studied. There are some differences observable in the process as transacted in the heart and in the voluntary muscles respectively, such as to require a separate description in each of these parts. And, first, of fatty degeneration of the heart.

Our knowledge of this disease does not run very far back. Frequent notices, indeed, occur in the older writers of sudden deaths which one can scarcely doubt were due to this change in the structure of the heart; and there are recorded observations of pale soft hearts which one feels sure the microscope would have found to be fatty. But there were no microscopes then available, or they did not stand, as now, at the anatomist's elbow, and fat hearts then arrested the attention which should have been given to the fatty hearts which we have only recently learned to distinguish as such.

A full bibliography of the subject has been compiled by Dr. E. Wagner,* and the labours of British pathologists have not suffered at the hands of their generous fellow-workman. Some later additions to the list may be found in Blachez's† thesis on fatty degeneration in general. The English student will find in Dr. Stokes's valuable work‡ a complete summary of all that has been made out on this subject, and references to the writings of the pathologists of the Dublin school, with whom fatty disease of the heart has long been a favourite study. I need scarcely allude to the classical essay of Dr. R. Quain,§ which perhaps has contributed more than any other paper to spread the knowledge of this disease, nor to Mr. Paget's lectures,‖ where the pathological picture is put before us with all an artist's skill; and if I add a reference to my own writings¶ here, it is as my plea to be heard on a subject which is still a favourite with me, and still, I think, capable of further elucidation by such studies.

Fatty degeneration of the heart, strictly so called, occurs in

* 'Die Fettmetamorphose des Herzfleisches.' Gr. 8vo. Leipzig, 1864.
† 'La Stéatose.' 8vo. Paris, 1866.
‡ 'The Diseases of the Heart and the Aorta.' 8vo. Dublin, 1854, p. 302.
§ On Fatty Diseases of the Heart. 'Med.-Chir. Trans.' vol. xxxiii. p. 121.
‖ 'Lectures on Surgical Pathology.' Second edition, p. 94.
¶ 'London Medical Gazette,' 1849, vol. ii. p. 739; 'British Medical Journal,' 1864, vol. ii. p. 152; and 1865, vol. i. p. 475.

two forms:* viz., a circumscribed and a diffuse form; the first being a chronic, the latter generally an acute form of disease. A third division may conveniently be added, which anatomically might be said to be allied to the circumscribed form, but pathologically takes its place among the most acute affections of the heart.

1. In the first form the disease occurs in minute pale specks chiefly found on and in the substance of the carneæ columnæ of the left ventricle. Sometimes the whole lining of the ventricle may be mottled with little spots of this kind seeming to run in zigzag lines; and sometimes they are observed beneath the pericardium. But their most familiar seat is in the musculi papillares of the left ventricle. They are found in connection with debilitating influences, such as hæmorrhage, long continued cachexia, and habitual intemperance. The circumstances under which they occur allow the disease to go on to produce those further changes which in the more widely diffused forms are necessarily interrupted by an earlier death. Within the narrow limits of these little specks the process may be traced from beginning to end; though, as these changes require time for their development, it is not in the same specks, nor always in the same heart, that the complete history can be followed out. The pale zigzag spots, the appearance most frequently displayed, belong to the later stage of the process where actual fatty infiltration has taken place, and perhaps atrophy of the fibres subsequently ensued. The indications of the early stage, of the actual commencement, are to be sought for in the little purpurous spots which dot the interior of the left ventricle in fever, hæmorrhage, phthisis, and other such-like exhausting diseases. Sometimes these spots are no more than what they appear to the unassisted eye, namely, mere ecchymoses beneath the lining membrane. At other times we may trace within these narrow limits the anatomical history of the lesion. In the muscular structure of the heart of a poor girl who died of pyæmia—to quote a carefully observed instance of this change in its minuter manifestations—were red patches, looking like ecchymoses, of a deep purple mixed with grey in the centre, becoming gradually paler at the edges. In the grey part there was a mixture of pus and blood, with fragments of muscular fibres. Surrounding this was a zone, in which the muscular fasciculi had lost all traces of striæ both transverse and longitudinal, they were simply granular all over. Then, for a space,

* Wagner, op. cit. p. 9.

the fasciculi were longitudinally striated, just as is seen in degeneration of the voluntary muscles. Outside this zone the muscular structure presented its normal appearance.

2. In the second or intermediate form the muscular structure is affected over a much wider extent. It seems to commence deep in the muscular structure, the wall of the left ventricle being affected by preference. As it spreads it approaches nearer the inner than the outer surface, involving whole layers of fibres in its progress, and following the direction of the fasciculi. The muscular fibres thus affected are of a bright yellow colour, so bright as often to give the idea of purulent infiltration. However, there is no purulent infiltration here; there is neither pus nor fatty matter in these discoloured patches, the yellow appearance being entirely due to decoloration of the muscular tissue. After a short exposure to air the bright yellow colour fades, and it is not long distinguishable in preparations preserved in spirits.

These broad patches are separated from the surrounding healthy structure by a deep red zone, the existence of which here, as in the form just described, marks an early active stage in the process of degeneration. We do not find the buff coloured spots in the interior of the carneæ columnæ, where the advanced stage of the disease leads us to infer that it has been long in progress, thus encircled. But it is round the seemingly recent purpurous specks, and round the bright yellow patches where the disease is still in an early stage, that the zones are found. The line of demarcation is not a line of separation like that which surrounds masses of so-called capillary phlebitis in the spleen and kidney. At least I never have found any satisfactory evidence of such separation having been effected. It looks like a sign of the presence of inflammation, which yet, judged by its results, as we understand the term inflammation, we can scarcely call it. Thus much at least, however, we may infer from its presence, namely, that increased vascular action is necessary for the process of complete disorganisation which ensues within the limits circumscribed by this zone.

The causes of this form of the affection are peculiar. It is found in connection with inflammation of the heart, by whatever means this may have been induced. Rheumatic inflammation extending from the pericardium seems, though I have never actually seen this change on dissection under these circumstances, to be a frequent cause of it. Over-training and athletic exercises pushed to foolhardiness do mischief in this way,

and so, more rarely, does overwork accompanied by misery and privation.

3. In the third form we find the affection generally diffused over the whole organ, which is then of a dull fawn colour, soft and rotten, moulding itself on the hand like a wet glove. In connection with this general change we may often find the familiar traces of the first form of this disease dotting the interior of the left ventricle; and habitual intemperance is a condition common to both these forms. This form, as a rule, is the anatomical expression of that peculiar cachexia, whether of old age or of less advanced years, which centres in and is often limited to the heart. It owns no recognised cause, and yields to no known method of treatment. Sometimes it is betrayed by the general signs of pallor and debility and cachexia during life. It is more frequently only discovered after, and as the explanation of sudden death.

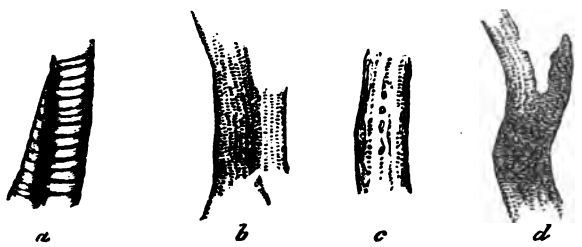
Proceeding to the more particular examination of the anatomical changes induced by the disease, we must observe, at the outset, that the transverse striæ of the muscular fibres of the heart are not, in the normal state, so distinct as those of voluntary muscle. They are placed, too, much nearer to each other, and they are more readily effaced by the use of glycerine in their preparation for the microscope.

In the commencement of fatty degeneration the striæ become less distinct, breaking up into rows of dots or granules, which, transverse in the first instance, in a somewhat more advanced stage affect the longitudinal direction. This appearance of granular disintegration is induced by other influences besides fatty degeneration. It is due to disintegration or disruption of the various elements contained in the primitive fasciculus.* It may be induced in the living body by inflammation, and after death it accompanies commencing decomposition, or attends on the action of certain chemical agents.

Commencing decomposition simulates very closely that change in the heart's muscular structure which is known as the first or granular stage of fatty degeneration. I have been deceived by it myself; and such a mistake is all the more likely to occur, because granular degeneration, like decomposition, may involve the heart uniformly over a very wide extent; much more widely than distinct fatty degeneration ever does, probably because the destruction of the muscular fibres is less complete.

* Todd and Bowman, 'Physiological Anatomy,' vol. i. p. 152. 8vo. London, 1845.

The pathological process is most perfectly imitated by prolonged maceration in dilute alcohol. By this the sarcous elements are disintegrated along the longitudinal lines of cleavage. By prolonged maceration in dilute nitric acid, disintegration is effected in both the longitudinal and transverse directions, and the fasciculus is resolved into small squarish powdery fragments. By varying these processes the results may be modified; and by such means the muscle may be disintegrated at will, in either direction and to any extent, independent of fatty degeneration. But, whether in the living or the dead body, and by whatever means induced, whether real or artificial, there is no increase of fatty matter in the product;



Changes induced in the muscular fibre of the heart in the course of fatty degeneration—

a. Healthy muscular fibre.

b. Granular degeneration, the transverse striation exchanged for longitudinal dotted markings.

c. Commencing fatty infiltration; the granular dots have been partially removed, and the centre of the fibre contains a few drops of oil.

d. Completed fatty infiltration and shrinking of the fibre where it is not distended by retained fatty matters.

All these figures are reduced to a scale of about 300 diameters, from drawings made by the aid of the camera lucida, with a one-eighth object glass.

the optical appearance is due to a mechanical change in the structure of the fibre. It is not a degeneration into fat, but a disintegration into granules—granular disintegration. Chemical analysis tells us distinctly what it is not, and microscopic examination is equally explicit as to what it is.

The granular fasciculi which result from the pathological process, or from any of these chemical reactions pushed only so far as to imitate it, require a high power to display the exact nature of the changes which have taken place. With a magnifying power of about 240 diameters only, it seems as if the transverse

striæ had been replaced by longitudinal rows of dots, divided here and there by other rows of larger and darker dots, and by fine lines. These appearances are explained by the application of a power of 450 diameters or more, which shows that the transverse striæ are not really lost. They are paler indeed than in the normal condition, but they preserve their normal distance from each other. The primitive fasciculus seems to be split into ribbons of unequal width by longitudinal fissures interrupting the transverse striæ. In consequence of these interruptions, such striæ as catch the eye with a lower power seem to run into rows of dots, and this false impression is heightened by the rows of larger and darker dots which occupy some of these longitudinal fissures.

The examination of this altered structure of the heart is much facilitated by the use of glycerine, according to Dr. Beale's directions. Unfortunately, this medium, so very useful in unravelling the fasciculi, greatly obscures their transverse markings. But this is remedied, to some extent, by the application of ether, which dissolves away the large dark dots in the fissures, and brings the transverse striæ again into view, drawing them at the same time nearer to each other, so that eight now occupy the space where only six were found before the ether was applied. Without the use of ether, the transverse striæ may be brought again into view by means of a condenser and a narrow stop. As the various objects in the field come out more distinctly, some longitudinally striated fasciculi may perhaps be recognised among the degenerated fibres; and from the ends of some of these which have been broken during the manipulation, ragged filaments may be seen projecting, just as we shall see presently in degenerated voluntary muscle. These filaments vary in length and breadth, but all have the transverse markings; some are broader; some are so fine that they might be fairly said to represent the old so-called ultimate fibres of muscle, their dimensions scarcely admitting of the presence of more than a single row of the component granules.

In the next stage all traces of transverse marking are lost. The few granules which still affect any regular arrangement are disposed in the longitudinal direction; they are no longer of uniform size, and oil-globules are to be seen mixed with the other molecules. Besides the optical evidence of the presence of minute oil-globules which have, at this stage, infiltrated themselves among the disintegrated sarcous elements, and of larger oil-drops which crowd the field of view, we now have

chemical proof that a new element has been introduced into the mass of tissue, and that the nitrogenous constituents have been replaced to this extent by fat. Not by margarine, the fat which is to a great extent characteristic of the human body, but by oleine, which is the fat of old age, the fat of disease.

The shortness and brittleness of the fibres which have undergone this change are qualities not very obvious under the high powers which are most conveniently employed in the enquiry, but they are nevertheless very important in relation to the connection which exists between rupture of the heart and fatty degeneration. Unnoticed, perhaps, in an ordinary microscopic examination, it is obvious enough, when we attempt to trace a fibre for any considerable distance, that fatty or granular fibres cannot be traced out with the same facility or to the same extent as healthy ones. By this mode of proceeding, we also find that a fibre is not always uniformly affected along its whole course, but that it may be granular in one part and fatty, or perhaps healthy, in another. Sometimes the outer part of the fibre may still display traces of the normal structure, while a row of oil-globules runs up the centre. The fact of the development of muscular fibres proceeding from more than one nucleus would lead one to expect that their decay might also proceed simultaneously from more than one point.

The third stage of this process is, as has been already remarked, more often reached in the circumscribed than in the more diffused forms. At this point the regular arrangement of the internal structure ceases to be distinguishable. At the same time, the fibre loses its symmetrical outline, swelling up in the parts where it is permanently distended with opaque fatty matter; or, where this is not the case, dwindling away. At last, a bundle of irregular threads, or the appearance of a cicatrix, may be all the evidence of the former occurrence of fatty degeneration which has passed through all its stages and gone on to its natural termination in obsolescence of the affected fibres.

We must not confuse this obsolescence, which is the ultimate result of fatty degeneration, and, as such, always of very limited extent, with that general induration of the muscular structure of the heart to which the term fibrous degeneration is, perhaps, more commonly, though less accurately, applied. This is an obsolescence, and betrays, in the irregular shrunken elements of which the resulting structure is composed, the history of its origin. Such a structure is merely

the remnant of the frame which has collapsed on the decay and removal of the parts which it held in connection.

The other process is the substitution of one healthy tissue for another in obedience to the altered requirements of the organ. The fibrous tissue in such indurated hearts replaces a certain proportion of the muscular tissue over a large extent, perhaps over a whole auricle or ventricle. It is found chiefly, but not exclusively, in connection with mixed hypertrophy and dilatation, affecting by preference the thinner portions of the heart's walls. The muscular tissue, under these circumstances, becomes hard and elastic; the cavity retains its shape when cut open; the muscle creaks under the knife; and the cut surface looks paler than natural, but of a white rather than a yellow hue. And under the microscope the explanation of these changes appears in the substitution or the addition of an unusually large amount of fibrous or fibro-cellular tissue.

In the heart this fibrous adventitious structure is employed to resist dilatation; it is the alternative of hypertrophy, the substitution of a mechanical for a vital force. The same substitution is occasionally found in the neck of the pregnant uterus. But the differing functions of the heart and of the uterus lead to different results from the presence of this misplaced tissue in the two organs respectively; and in its occurrence, I believe, lies the explanation of that terrible accident, rupture of the uterus during labour. As far as I have seen, a ruptured uterus does not give way from rupture of fibres which have undergone fatty degeneration; indeed, I am not aware that this form of disease occurs at all in the pregnant uterus. It is ruptured, not at its weakest, but, mechanically speaking, at its strongest point, the torn edges displaying strong fibrous tissue, and not degenerated fibres.* On the other hand, it appears that hearts which have undergone fibrous transformation are not liable to rupture. But, while this change resists dilatation most efficiently, at the same time it limits the contraction of the chamber of the heart thus involved, and reduces it so far to the condition of a mere containing vessel.

Fatty degeneration of the voluntary muscles is essentially the same process as fatty degeneration of the muscular structure of the heart. But there are certain differences, most strikingly shown in the ultimate results, referable to differences in the functions and the anatomical structure of the two fibres

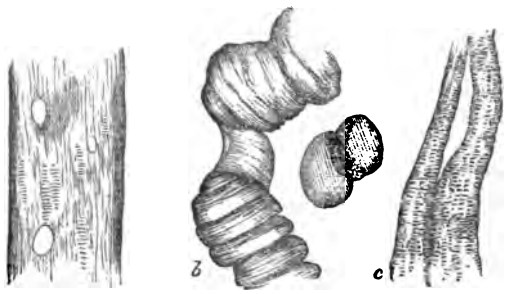
* See some observations on the subject in the 'Lancet' for 1860, vol. ii. p. 306.

respectively. None of the pathological differential characters are absolutely foreign to the muscular tissue of the heart, but some of them are very rarely met with and indistinctly seen there, and can only be thoroughly understood by studying them in their more usual seat, and in their fuller manifestation, in the voluntary fibres. The necessity for a continual action of the heart for the maintenance of life, which affects the process and ultimate results of fatty degeneration of this organ, does not apply to the voluntary muscles, and in these, accordingly, the process more often goes on to its natural termination. In these ultimate results, the anatomical differences of the two kinds of fibre are to be traced.

We may observe, in the first place, that, in a voluntary muscle in which this change has gone on to the extreme degree, there is as much fatty accumulation as fatty degeneration. The pale yellow muscles which we find occasionally in sheep's backs or in the forearms of the subjects of lead-palsy, supply the best illustrations of this. The most readily-accessible illustrations, however, are supplied by the muscles of limbs atrophied in connection with disease of the joints. If we tear up a fragment of such a muscle under the microscope, we find that the chief bulk consists of fat, which is deposited in lines or strings running in the direction of the fibres; not replacing them, but lying in their interspaces—lying, in fact, just where fat lies in healthy, well-fed animals. Between these lines of fat, and occupying much less space than they do, we find muscular fibres in all conditions—from perfect health to the most extreme degeneration. And, besides, bands of fibro-cellular tissue occur, which add very much to the difficulty of the dissection. Such, and such general fatty degeneration as we find in the heart occurs very exceptionally in voluntary muscles.

The first stage of fatty degeneration of the voluntary muscles, as far as I have been able to satisfy myself, is characterised by swelling of the affected fasciculi. For a greater or less length they appear uniformly dilated to about twice the size of the healthy fasciculi, smooth on the surface, seemingly wanting the transverse striæ, but streaked longitudinally with fine linear markings running the whole length of the fibre. These may not be very distinct in their even course, but where the fibre has been stretched, or twisted, or broken across, they come out quite clearly. Sometimes they are gathered up into a point like the end of a camel-hair pencil; at other times the broken fibres look like the unravelled ends of a rope with the

strands hanging out, having undergone at these points complete longitudinal disintegration. A higher power shows here, as in the muscular structure of the heart, that the transverse striæ still exist, only they are much paler than natural. They are not interrupted by the longitudinal markings. They are approximated to half the normal distance, sixteen of them occupying the same space as eight do in a healthy fibre. Ether does not cause them to approximate more closely, though it seems to shrink up the fasciculus; and it displays the longitudinal markings very clearly, at the same time that it extracts some fatty matter from its interior.



Changes induced in voluntary muscular fibre in the course of fatty degeneration:—

a. Swelling of the fasciculi with longitudinal streaking. The transverse striæ approximated where still visible.

b. Transverse disruption of the swelled fasciculi. Two fragments have separated entirely from their original connection. The articulated masses are held in connection by an empty string of thickened sarcolemma.

c. Granular disintegration with atrophy of the fasciculus.

These figures were drawn in the same way and to the same scale as those of the changes in the heart's fibres, but they are here represented enlarged only about 150 diameters.

This longitudinal cleavage would seem to hold nearly the same relation to fatty degeneration of voluntary muscle as granular disintegration bears to that of the heart. Nearly, for it passes more directly into fatty degeneration than the corresponding change in the heart does, ether readily extracting oil from the streaked fasciculi. And it is more specifically a pathological process, being much less readily imitable by any artificial reactions. For chemical decomposition and putrefaction do not induce this particular appearance, but seem, as far as I have observed, to pass over it to a further stage, converting the fasciculus at once into a finely granular mass, which

retains only the outline of its former structure. This process of conversion may be traced in some of the transverse striæ which have survived the rest. These appear distinctly granular, broad, and uneven; as if falling to pieces under the action of the dilute nitric acid which has disintegrated the sarcous elements elsewhere. The striæ seem to persist longest just beneath the sarcolemma. Some fasciculi which appear granular owe this deceptive appearance to the striæ on the two opposite apposed sides of the flattened fasciculi crossing each other diagonally, the regular arrangement persisting on the surface for a while after the sarcous elements in the interior have been deranged or have been dissolved entirely away.

Sometimes these large pale waxy-looking fibres occur alone. More commonly, however, we find mixed with them round or oval masses, which seem, from their general appearance and dimensions, to have originated in the breaking up of the longer fibres. These may be either striated longitudinally, or minutely granulated, like the fibres from which they seem to have originated by a transverse fracture of the brittle fibre. This transverse cleavage is displayed most strikingly in fragments of a very peculiar form which are rarely met with. It was from the neighbourhood of an aneurism that I have been supplied with the best specimens of muscle which had undergone this very peculiar form of disintegration. The transverse divisions were in this instance so very regular, and the appearance was so unlike muscle and so like muscular entozoa, that it was only on Dr. Beales's corroboration I ventured to conclude that the masses in question were, or rather had been, really muscular.

These are neither granulated nor streaked longitudinally, but they are cracked and broken transversely, and they are often connected at either end, as if they had but partially escaped, with a string of sarcolemma. They have a singular affinity for the colouring matter of carmine, which tinges them more deeply than any of the other structures which the altered tissue presents. Though they are found under the same circumstances as, and in connection with, the pale waxy-looking fibres already described, yet I am not sure that this transverse splitting is one of the series of pathological changes which lead to fatty degeneration. I am, as yet, uncertain whether these fragments, with the rounded granular masses and the more enigmatical articulated forms, do not melt away without undergoing any further change.

Coincidently with the disintegration of the sarcous elements

the investing membrane of the fibre is thickened. It may be traced in its passage from one wrinkle of the fibre to another, stretching across between two portions which have been displaced, or, perhaps, lying in folds, like a loose stocking, where the fibre has been pressed in at the ends. The thickening of the sarcolemma is accompanied by the appearance of numerous irregular fragments which cloud the field of the microscope. Some few of these are the nuclei of the muscular tissue; others, and these the most numerous, are apparently the rudiments of fibro-cellular structure; others are of uncertain nature; and many are undoubtedly collections of oil-globules, arranged like rows of squarish beads. These last are removed by ether, which clears the field a good deal, while acetic acid has very little effect in this way. And with these are a number of larger loose oil-globules, which have been set free during the manipulation of the tissues.

As in granular disintegration of the fibres of the heart, so in that of voluntary muscle; the longitudinal markings, which have replaced the transverse striæ, are in their turn obliterated. Dots appear, first in regular lines, then without order; and the total disruption of the sarcous elements and all their subsequent changes follow in the order which has already been traced, and might be described in the same words as have been employed to tell the complete history of fatty degeneration of the heart.

As in the muscular fibre of the heart, these preliminary changes occur in connection with other processes besides fatty degeneration, and notably with any local irritation. Like granular disintegration of the heart, they indicate a condition which may terminate in fatty degeneration, but in which, thus far, no fatty degeneration, no change of the chemical composition of the structure has taken place. Here, as in the heart, the results of commencing decomposition are, as far as we can trace them, identical with these organic processes. Here, as in the heart, is a common point from which the two series of changes—the chemical and the pathological—diverge to their respective terminations. And, before dismissing this branch of the subject, it will be of interest to point out some of the various pathological conditions which tend to this common point.

The changes which we have just been considering as preliminary to fatty degeneration are identical with those which Dr. Zenker* has described, and most beautifully delineated, as

* Zenker, 'Veranderungen der willkührlichen Muskeln in Typhus abdominalis.' Leipzig, 1864.

occurring in the voluntary muscles in the course of typhus fever. The pale swelled fibres with linear markings, the rounded granular fragments, the scattered nuclei and embryonic connective tissue, as displayed in his plates, might be taken as exact illustrations of the stages of fatty degeneration. The same familiar forms appear again in Dr. Bennett's* figure of the changes induced by cancerous tumours in the muscles in their immediate neighbourhood. But perhaps the most interesting observations on this subject are those made by Mr. A. Stuart,† of Petersburg, detailing the results of an enquiry into the effects of inflammation on living muscle.

By the application of nitrate of silver to the muscles of frogs, changes were induced in them identical with those which we have just traced in the heart. The parts directly exposed to the chemical action of the caustic swelled or shrunk up, and their texture was destroyed. But those a little further off underwent a regular series of changes, of inflammation in fact, which Mr. Stuart describes with great minuteness. Thus, briefly, first the muscle became pale and more transparent than natural; then the sarcous elements seemed to break up and arrange themselves in longitudinal rows; then these dots became larger, though still remaining sarcous elements. These changes occupied two or three weeks, the process thus far being disintegration rather than degeneration, no fat having as yet appeared in the altered fibres. Then the last stage set in, namely, the conversion of the albuminous substance into fatty matter.

The conclusions which seem fairly deducible from these observations on fatty degeneration of both kinds of striated muscle may be expressed in a few sentences.

Inflammation, anæmia, cachexia, want of nutrition, and all the other causes to which fatty degeneration of the heart has been ascribed, have thus much in common, that they disorganise the fibre and prepare it for ulterior changes. The fibre is functionally destroyed by these means, and the disintegration is a change which ensues in the disorganised tissue.

The first step which we can recognise in the process of, or towards, fatty degeneration of muscular tissue, is longitudinal or transverse disintegration of the sarcous elements into filaments, or discs, as the case may be. Circumstances will determine which of these forms the disintegration shall take, and, probably, the exciting cause of the disease has a certain

* 'On Cancerous and Cancroid Growths,' p. 105, fig. 121.
† Schultz's 'Archiv für microscop. Anatomie,' i. S. 415.

influence in this particular. But, although the exciting cause may have a modifying influence, the general course of the degenerative process is guided and transacted by the ordinary agency of the living body, irrespective of these partial influences.

The actual fatty degeneration is secondary in every sense to this disintegration. Its occurrence depends on the existence of fatty matter in the blood available for this purpose. And it is conceivable that, for want of an available supply of fatty matter, disintegration should not be followed by fatty degeneration at all. If this be true, a question suggests itself, whether this explanation be limited to fatty degeneration of muscular tissue, or whether it has a wider bearing. The answer to this question must be sought in the examination of fatty degeneration of other tissues.

Fatty Degeneration of the Liver.

Fatty degeneration of the liver supplies at once the most familiar and convenient illustration of this form of disease as it affects glands. There are difficulties in the investigation, arising partly from the complexity of the organ, and partly from the fact that fatty infiltration is, to a certain extent, a normal condition of the hepatic glandular structure. But we have nothing to do with these sources of difficulty just now. For, in the first place, we are, from the present point of view, only concerned with the proper secreting structure. When fatty degeneration of the liver is spoken of, unless some other structure is specially named, it is generally understood to denote fatty degeneration of the glandular epithelium which fills, in whatever way, the lobules of this organ. Again, we have not here to determine the precise point at which fatty infiltration of the hepatic cells passes the physiological limits and constitutes a morbid change. For it is only when the nature and degree of the change is quite unequivocal that it comes within our present scope. These questions, all-important in considering the history of fatty degeneration of the liver in itself, have but a secondary importance in relation to the subject of our present enquiry, namely, the general pathology of this affection.

As in the heart, so here in the liver, fatty degeneration may pursue its course in different ways, and with different degrees of rapidity. We may find general fatty infiltration pervading the whole organ, in a form whose progress we measure by

months or weeks, and which we connect with excessive absorption of fat from the alimentary canal or with its defective elimination from the lungs. Or we may find the results of a more chronic change, where we date by years or months, and where the history of the disease may be traced in the successive stages of its progress, as displayed in different parts of the organ. And yet again, though more rarely, we may find in a very unusual form of disease, known as acute yellow atrophy of the liver, a clue to the general pathology of fatty degeneration of this as well as of other organs. The diffused form of fatty degeneration of the heart finds a parallel in the changes which occur in the liver. And so, too, though the total amount of the changes which the heart displays, under the circumstances, falls far short of those which cirrhosis induces in the liver, does that circumscribed form which runs on uninterrupted to its termination in the carneæ columnæ of the left ventricle. Nor, as we shall presently see, is acute yellow atrophy of the liver a change so entirely sui generis. The mode of its extension and its results are strictly comparable to those of what we have already described* as a special form of affection of the muscular structure of the heart.

Fatty degeneration of the liver, as familiarly known, appears, in the first instance, to be merely an increase of the amount of oily matter which is found in the physiological state in nearly all the hepatic cells. The gland-cells at the outer edge of the lobule are chiefly thus affected. The oil-globules may be larger as well as more numerous than those which occur in health, but this does not seem to impair the functional activity of the cells which contain them. For these cells still retain their nuclei, and still, mostly, contain bile; and, doubtless, many a liver advances thus far in the appearance of disease, and then returns within the physiological limits.

When the change is more widely extended, involving the lobules more deeply, as in some cases of pulmonary phthisis and in the cachexia of drunkards, we cannot doubt that we have to deal with something more than a temporary infiltration of fat. We find now a large number of loose oil-globules, which have been set free by rupture of the hepatic cells that had contained them. And in a large proportion of cells no nuclei are to be seen. These cases, however, illustrate only one stage of fatty degeneration, and give less insight into its anatomical history than do those where the affection has been more

* Page 42.

partial and less rapid in its progress, such in fact as cirrhosis supplies.

In the next stage, so abundantly illustrated in the dissecting room of every hospital, the fatty infiltration may seem to have changed its situation. It no longer appears in zones corresponding to the capillaries of the portal circulation, but in the form of little opaque ochrey spots, surrounded by rings of clearer pale tissue. The microscope tells us that these opaque yellowish specks owe their peculiar appearance to the presence of numerous hepatic cells which are loaded with fatty matter and stained with bile. These cells are unusually coherent, and the fatty matter which they contain is not, I think, so easily soluble in ether as that which is found nearer the edges of the lobules in an earlier stage of the affection. The proportion of cells which have no visible nuclei is constantly on the increase, with the advance of the disease. The pale somewhat transparent tissue which surrounds these opaque yellow specks seems to be composed of a fibro-cellular substance containing the vessels, now shrunken and obsolete, but no trace of the glandular epithelium of the original structure.

The history of the process seems complete, as gathered from different specimens representing these successive stages. Apparently the process of degeneration has travelled from the circumference of the lobules to the centre. First the cells became permanently infiltrated with fatty matter; then they were shed, and none were produced in their places; while the space which they had occupied—the cavities, if I may assume thus much, in which they grew—collapsed. And a tough fibro-cellular tissue resulted, traversed by shrunken vessels suggestive of the cause of the portal obstruction and of the dropsy by which the last stage of fatty degeneration of the liver is so commonly attended.

The mutual connection of these changes is very distinctly shown in the form of disease already alluded to as acute yellow atrophy of the liver.* For the most part this is an acute form of disease effecting the disorganisation of the liver in a few days, accompanied by fever and delirium, and characterised by the brightest yellow jaundice. In such cases few or no hepatic cells are to be found in the parts of the liver which the disease has invaded—perhaps the whole of the organ—only loose oil-globules and particles of bile. These cases, however, are less intelligible, and less applicable to our present purpose than those where the progress of the disease has been slower. For, under

* Rokitansky, 'Path. Anat.' ii. S. 313, 2te Aufl

such circumstances, as the longer period allows, the line of demarcation between healthy and diseased tissue is more strongly drawn, and the successive stages by which the final disorganisation is reached are more clearly to be traced. To this less common form of the disease I wish particularly to call attention.

The healthy tissue, in this form, is represented by bright yellow round nodules easily separable from the rest of the organ, some appearing even to be encysted, on account of the consolidation of the interlobular connective tissue which surrounds them. If the yellow colour is not apparent at first it is brought out on exposure to the air, when the whole surface appears mottled with bright yellow and deep purple in the strangest manner. These yellow nodules contain hepatic cells, which are altered more or less according to their distance from the centre of the lobule. As in the commoner forms of fatty degeneration, the disease advances from without inwards. As we follow the steps of the pathological change we notice first that the cells become granular. They contain a little oil, but certainly in no excessive quantity; there is perhaps even less than in the normal condition. The biliary matter, on the contrary, seems to accumulate within the affected cells. The cells lose all tendency to cohesion to each other, and, where the disease has advanced farthest, they part from the basement membrane also; for in the circumference of the lobules no cells whatever are visible—they seem to have dropped off, and to have been dissolved or discharged.

The present seat of the disease, the tract within which these changes are proceeding, is indicated by a deep purple stain, while the parts which have not as yet been involved are in a state of biliary congestion indicated by a bright yellow colour. This congestion, however, means no more than puerile respiration does under parallel conditions of the lungs, namely, that a part is undertaking the duty of a whole organ. The purple stains are wider in proportion as the disease is more active and its progress more rapid. In the chronic forms these are narrowed down to thin lines and rings, which separate the parts still capable of performing their functions from those which have been disorganised. The microscope finds in these purple patches few or no oil-globules; a few hepatic cells which may possibly belong to the yellow part; and a few small granular masses of very variable size; but no pus corpuscles or large compound granular cells. The line which, as it were, separates the living from the dead is, in the liver, as in the heart, the seat of excessive vascular congestion. Inflammation may ensue, as a secondary

consequence of the local action, showing itself by the formation of adhesions along those lines where they happen to crop out on the surface of the organ. But there is no reason to consider the disorganising process as essentially inflammatory.

The parts where the process has gone on to complete disorganisation differ in appearance according to the length of time that may have subsequently elapsed. In recent acute cases they are dark, much below the level of the surrounding tissues, and of a soft yielding texture. In chronic cases, where the history gives us reason to believe that a longer time has intervened since the invasion of the disease, they are collapsed and pale, and, though flexible, they resist the knife like the tissue of a cicatrix. The microscope here, as in the results of ordinary cirrhosis, shows nothing but a fibrous structure in which the course of the different vessels can still be traced, the epithelium cells having been all shed, leaving nothing to indicate that we have under examination what was once glandular tissue. In one case of several months' duration, where death had been preceded by all the usual signs of portal obstruction, the small bloodvessels in the interlobular spaces were convoluted like the vessels in the spermatic cord. A few oil-globules lay in this disorganised tissue here and there, but no hepatic cells could be seen anywhere in it.

Fatty Degeneration of the Kidney.

The analogy between fatty degeneration of the kidney in its different forms and fatty degeneration of the liver is very close. The different mode of arrangement of the glandular tissue in the two organs respectively involves certain differences in the disposal of the effete products of the disease. The secondary constitutional results, too, vary, of course, according as urea or bile is retained in the system. But where these do not interfere, the identity of the disease in the two organs is almost absolute.

We owe our knowledge of this subject to the labours of Dr. G. Johnson.* Venturing to draw from his observations conclusions somewhat different to his own, I feel that I could have no greater security from being led astray by my preconceived notions than to adopt his classification and his excellent descriptions in detail, and I have reproduced them accordingly. Further, never having made this class of diseases of the kidney

* 'On Diseases of the Kidney' and 'Med.-Chir. Trans.' vols. xxix., xxx., xxxiii.

the subject of particular examination, I gladly avail myself of the opportunity of testing the correctness of my views by his independent observations.

Fatty degeneration of the kidneys occurs in two forms. In one the kidneys are large, smooth, soft, and mottled. 'The convoluted tubes are found to be everywhere greatly distended with oil, which has accumulated in their epithelial cells. The detached epithelial cells, when filled with their oily contents, have often a remarkable resemblance to the cells of the liver in the same condition. The fatty accumulation occurs in the epithelial lining of those portions of the tubes whose office is the secretion of the solid constituents of the urine. In some of the tubes the epithelial cells cannot be seen; they appear to have undergone a process of atrophy, and the tubes are occupied only by their oily contents.'* 'It is this form of fatty degeneration of the kidney which occurs in animals, as a consequence of confinement in a dark room.'†

In the other form, also, the kidney is generally large and soft. As time goes on it may shrink, and in such case become harder. But the character which marks this form of the disease is the uneven granular surface of the organ. 'In the granular kidney, many of the tubes contain a number of detached oily cells, which are readily washed out by the stream of urine passing through the tube; while in the mottled kidney there is commonly only a single layer of epithelium, which still remains adherent to the basement membrane.'‡ 'The greater part of the oil is contained in distinct cells; and these can readily be traced through a gradual series of transitions up to the gland cells of which they are, evidently, a degenerated offspring.'§ 'The convoluted tubes are found filled in different degrees with oil, some tubes being quite free, while others are ruptured by the great accumulation in their interior. The opaque yellow spots scattered throughout the kidney are convoluted tubes distended, and many of them ruptured by their accumulated fatty contents; just as the red spots are convoluted tubes filled with blood. In parts of the same kidney, there may commonly be seen some of the appearances already described as indicative of desquamative nephritis.'‖

The analogy of these two pathological conditions of the kidney to general fatty infiltration and cirrhosis of the liver

* 'On Diseases of the Kidney,' p. 388.
† 'Med.-Chir. Trans.' vol. xxx. p. 183.
‡ Op. cit. p. 391. § Page 384.
‖ 'Med.-Chir. Trans.' xxx. p. 183.

respectively are sufficiently obvious. I am not aware of any affection of the kidney which could be strictly compared to acute yellow atrophy of the liver. For, although the acute desquamative nephritis which sometimes follows scarlatina presents some points of comparison, yet the power of repair which the kidney possesses affects the resemblance in a most important point. In the kidney the glandular epithelium is reproduced, and the organ is restored to perfect integrity; while in the liver, as we have seen, the secreting structure is not restored, and the organ collapses. Stripping of the glandular lining of the renal tubes and subsequent—consequent—collapse of the organ does, however, occur, as a result of what Dr. Johnson calls chronic desquamative nephritis, in the small contracted kidney. In the cortex of one of these kidneys the appearances described as occurring in the portal zones of a cirrhous liver are reproduced. The framework is there, the vessels and tubes and the hard connective tissue, but the gland tissue is gone, and the empty tubes lie collapsed where they are not distended by effete epithelial cells.

I believe that these independent observations on the kidney confirm in every particular my own conclusions from the examination of the liver. Though, in the absence of such a test as acute yellow atrophy supplies, the renal could scarcely be made the basis of such a general inference as the hepatic changes. Both alike seem to show that fatty degeneration is not the first step, but a secondary affection of disorganised cells. If the cells are thrown off simultaneously with their disorganisation, and pass away, there will be no evidence of fatty degeneration at all. But if retained, whether free in the cavities of the gland, as in the granular, or still adherent to the basement membrane, as in the mottled kidney, they will be infiltrated with oil. Lastly, these observations on the kidney show that, after the cells are shed, the cavities which contained them collapse, and the organ, to this extent, undergoes the same retrograde changes as obsolete structures display everywhere else.

Fatty Degeneration of the Placenta.

Fatty degeneration of the placenta has been so fully described* that it might seem almost superfluous to open the subject anew. But, after a careful anatomical examination of

* Barnes and Hassall On Fatty Degeneration of the Placenta, 'Med.-Chir. Trans.' vols. xxxiv. and xxxvi.; Druitt, 'Med.-Chir. Trans.' vol. xxxvi.; Kilian, 'Brit. and For. Med.-Chir. Rev.' vol. vii. p. 272; Handfield Jones, 'Path. Trans.' vol. iv. p. 238.

the whole subject, I must venture to express my dissent from some of the conclusions which are generally received with regard to this pathological condition. I would suggest, as the result of my observations, a somewhat different arrangement of the description of the various anatomical changes, and certain qualifications of the inferences which have been deduced from them.

Few human placentæ are entirely free from a peculiar local change, which shows itself in the form of hard white lobular patches, chiefly affecting the edge of the organ and its maternal aspect. In the collapsed state in which we find this organ after delivery, they are sensibly elevated above the surface. A vertical section of one of these patches shows the change of colour and consistency to extend from the surface into the substance of the placenta, to a greater or less depth, sometimes through the whole thickness of the organ. As we trace its lateral extension, we find the lobules of the placenta to coalesce into broad hard white tracts where few or no pervious blood-vessels are to be seen, and in which the proper functions of the organ must have been entirely suspended. Sometimes the change seems to have begun on the fœtal surface, and occasionally it would appear as if the fœtal and the maternal surfaces had been affected simultaneously, a line of comparatively healthy structure separating the confines of the two tracts of disease. In these white patches the evidences of fatty degeneration of the placenta are to be sought for.

Not every white placenta, however, is fatty, for the organ may be blanched throughout its entire extent, merely from the fact of its having been drained of blood, in the course of, or as the cause of, a miscarriage. Not even every placenta which displays to the unassisted eye the familiar broad white patches just described is fatty. For the disorganisation which is indicated by these obvious characters may be of more than one kind, to only one of which the name of fatty can be properly applied. Two of these I propose to describe as the typical forms of placental degeneration, the fatty and the fibrous, as chiefly displayed in the villi of the chorion.

In the first form, these patches are of a loose texture; they are separated from the healthy tissue by no very distinct line of demarcation, and on pressure they give issue to a creamy fluid. By the action of ether, we may obtain unequivocal chemical proof of the existence of a fatty matter in abundance in the disorganised tissue, rather soft, but solid at ordinary temperatures. And, by the help of the microscope, we may

trace in these patches all the morbid appearances which have been so well described by the authors already alluded to.

These patches want the smooth hard surface which characterises the other form, to be described presently. On tearing them up with the dissecting needles, we find them to consist of villi closely packed together, so closely, indeed, that it is difficult to unravel any considerable length of tube continuously from the friable mass. The tubes are granular and opaque in parts: the glandular epithelium is indistinct, little oil-globules appear here and there, and the extremities and lateral diverticula of the tubes are, many of them, filled with opaque yellow granular matter. This may be cleared by a solution of caustic potash, but the colour and opacity reappear on neutralisation by acetic acid. It is a granular-fibrinous rather than a fatty matter. Little oil-globules are, however, distinctly visible on the walls of the tubes, and in a more advanced stage of the degeneration, larger drops may be seen lying within the cavity of the tubes, or loose among the tissues. As we recede farther from the maternal surface of the placenta, which is the apparent starting-point and chief seat of the change, the tubes gradually resume their normal characters; the ends and diverticula become clear, and the epithelial glandular edging—as it appears—of the canal is again visible.

In the other form, the change does not seem always so closely connected with the maternal surface of the organ as in that just described. The white patches, too, have characters of their own. They are hard and compact, and are separated from the healthy tissue by well-defined abrupt boundaries. They are smooth, almost polished, on the free surface. When they have been cut or torn, no creamy fluid exudes on pressure; and the microscope finds no fatty matter in them. The action of ether gives the same negative results. By agitating equal portions of one of the hard and one of the softer white patches in separate test-tubes with ether, and evaporating the solution, the fact may be placed beyond a doubt that the soft patch contains fatty matter in large quantity, and the hard one little or none at all. With pains and patience the villi of the chorion of a fatty placenta may be unravelled almost up to the maternal surface. We cannot, indeed, follow a single tube so far, but we may disentangle the soft friable mass, in some fashion, to this depth. In a typical specimen of the fibrous form under consideration, however, this is quite impracticable. Up to the edge of the hard white mass, as seen on a vertical section, the villi are quite healthy and of their normal size. But there they

suddenly shrink into threads, which can only be followed separately for a very short way. By the help of acetic acid, which clears, and somewhat softens, the hard opaque mass, the course of some of the tubes may be traced a little farther, rather by the eye than the dissecting needle, the arrangement of their nuclei, and the points where the section has crossed their direction, showing the lines of obsolete tubes. Near the maternal surface minute oil-globules are scattered here and there; and deeper in the centre of the mass a few may be seen, not scattered, but gathered into clusters, as if within some vessel. But the fatty matter forms no appreciable proportion of the bulk of the substance, whether examined by the microscope or by chemical reagents. The mass is almost wholly made up of obsolete tissue, apparently of collapsed, contracted, and mutually adherent villi. And its texture and appearance strongly resemble those of the tissue which we find in the interlobular spaces of the liver after the destruction of its glandular elements. Limiting the application of the term fatty degeneration to the form already described, I would designate this as cirrhosis of the placenta.

There are two forms of fatty degeneration, which I have not met with in the placenta, wanting to complete the series, so as to render the history of the disease here parallel to what we find in other organs.

I have not met with an instance of fatty degeneration of the placenta parallel to that form of the disease when a whole organ seems to be infiltrated with oil. Dr. Barnes * cites a case where fatty degeneration of the placenta occurred in a fat woman; and, as no disease appeared in the child, the change in the placenta was referred to some affection of the mother. I have myself seen just such another case. But it cannot be said that these differ in any essential point from other cases, except so far as the probable excess of fatty matter in the blood of the mother allowed the fatty infiltration of the placenta to proceed with greater rapidity than it would have done had fatty matter been less easily available. And it seems to me that a fatty infiltration of the placenta, strictly comparable to the fatty infiltration of the liver—to take this instance—which occurs in connection with tuberculous cachexia, would not take the form of local disease. It would probably have its seat in the cells of the decidua, as well as of the chorion generally, and not in certain parts only which had been prepared by previous changes to receive the fatty deposit. The physiological peculiarities of

* Op. cit. vol. xxxvi. p. 167.

the placenta render such an occurrence exceedingly improbable. Like general fatty degeneration of the heart, it could not go on far without involving the death of all that depended on the particular organ, and so cutting short the pathological process. And I do not find any evidence of the occurrence of this form of disease, in any of the cases recorded by Dr. Barnes or Dr. Druitt.

Another condition which I have also, as yet, failed to notice, is a change in the placenta parallel to that which accompanies acute yellow atrophy of the liver. Such a condition might perhaps be found in connection with abortion in fever, and be traced in the cells of the decidua and chorion.

The physiological peculiarities of the placenta are reflected in its pathology, and we cannot rightly interpret its morbid appearances without a full consideration of the normal relations of the different elements which enter into its composition. The chorion is not merely a frame on which the bloodvessels are extended, a means of adhesion between the ovum and the uterus: it is as truly a glandular structure as the liver or the kidneys. Not only is the fœtal blood depurated in its radicles by apposition to the mother's blood, but a supply of new material is constantly being elaborated by the layer of glandular epithelium which is spread over the decidua, as well as the chorion at the surface of contact. The placenta discharges the functions of the stomach as well as of the lungs.*

For this purpose, two separate systems of vessels are brought into mutual apposition during a certain portion of their course. From this point the umbilical veins bring back the blood depurated, oxygenated, and, as from other ductless glands, carrying in it the secretions of the placenta. If the maternal circulation fails at any single point, or if, as from a shock over the whole surface, the decidual glands fail to that extent in consequence, the fœtal elements of the placenta repeating the failure, the blood returns from that point, or that surface, as it came, neither depurated nor enriched with nourishment for the embryo; and then the corresponding fœtal portion of the placenta fails and shrinks up with the abolition of its functions. Conversely, the failure of the fœtal circulation at any point will be followed by the obsolescence of the corresponding maternal structures of that portion of the placenta.

On these grounds, the explanation of fatty degeneration of the placenta seems quite clear, and coherent in its analogy to that of fatty degeneration and its allied changes in other

* See Goodsir, 'Anatomical and Pathological Observations,' p. 50.

organs. An effete gland-cell exposed to the influence of passing blood containing oil will become infiltrated with oil. But if the interruption of the circulation be sudden, and with it the access of oil be cut off, there will be no fatty infiltration. The tissue will collapse, and cirrhosis, or fibrous degeneration, will ensue. This was well shown in a placenta which had been separated suddenly by hæmorrhage along a line a few weeks before delivery. It might seem as if the alternative between fatty and fibrous degeneration of the placenta were merely a question of the sudden or gradual interruption of the circulation in its vessels.

In relation to the general pathology of fatty degeneration, an argument may be derived from this part of the subject, which has been strangely overlooked by those who insist on the direct convertibility of animal tissues into fatty matter. And, perhaps, the same fact might be adduced as support of the opinion that fatty degeneration proceeds from the maternal, and fibrous from the fœtal, vessels. Merely this: that the tissues of a dead fœtus are not liable to fatty degeneration under any circumstances. The products of extra-uterine fœtation may remain enclosed in the living body for years without undergoing this change. The little embryo which is found in a mass of so-called uterine hydatids has undergone no fatty degeneration. And the fœtus which has lain for months and years in the uterus of a cow is found shrunk and blackened, but not converted into fat. This is quite in accordance with what has been previously stated. The dead fœtal tissues are removed from any supply of oil by which they could be infiltrated. There must be molecular apposition if not absolute continuity between the disorganised tissue which is to receive the oil and the living tissue whose moving blood is to supply it; though, as in the case of fatty degeneration of the cornea, an actual capillary continuity is not required.

If these inferences be correct, fatty degeneration of the placenta, such as has just been described, can scarcely be regarded as a change of any importance with regard to the condition of the fœtus. And, as a rule, large healthy children are born in connection with extensive degeneration of the placenta, having derived their nourishment from the portions which were not involved in the local change. Nor, again, can it be looked upon as having anything to do with one of the great perils of childbirth—adherent placenta. For the edge of the organ, where these patches chiefly occur, is just the part where adhesion of the placenta to the uterus is least frequently found.

On the contrary, from recorded cases, it might seem that these hard unyielding masses, already in such loose connection with the uterus, were more likely to induce a still farther separation of the placenta, and consequent hæmorrhage. Nor, lastly, can we regard it as the natural termination of the life of the placenta, for, certainly, this organ is not least active at the completion of the period of gestation. It is a local, not a general disease, a consequence, not a cause, and, interesting in the highest degree as a pathological study, is, I believe, devoid of all clinical importance.

I have had no opportunity of studying the changes induced in the cornea by fatty degeneration, as displayed in the arcus senilis, so well described by Mr. Canton.* So I will merely extract from his pages an account of this morbid appearance.

In the first instance, the corneal cells are the seat of the affection. These cells, in form comparable to the lacunæ and canaliculi of bone, lie between the lamellæ of the cornea. They are so numerous that their opacity imparts a cloudiness to the whole structure, though the lamellæ may not be affected. The superficial laminæ are first involved, and thence the change extends deeper into the substance of the cornea. Presently, the lamellæ themselves are changed, infiltrated by myriads of minute oil-globules, which, as they coalesce into larger drops and collect between the lamellæ, add notably to the thickness of the cornea. The opacity due to changes coincident with old age has much in common with that which results from injury and inflammation of this part.† They both consist essentially of infiltration of the corneal cells with oily matter, but, in the former the change is permanent; in the latter it is—that is, as far as it depends on oily infiltration of the corneal cells—remediable.

The term atrophy does not seem to me to express the correct pathology of this affection. Dr. His,† as cited by Mr. Canton, has put this very pointedly. Demurring to the opinion that the arcus is the result of simple atrophy, he asks, 'How is it that it is just in the margin of the cornea (which is in the most favourable position for nutrition) that such atrophy should occur; and how does it happen, in connection with such an atrophied margin, that the centre of the cornea can preserve its complete integrity through years? These are questions which at present appear to be quite insoluble.' I believe that

* Canton, 'On the Arcus Senilis.' 8vo. London, 1863.
† Canton, op. cit. pp. 30, 32. Virchow 'Cellular Pathology,' transl. p. 301-306.
‡ 'Histologie der Cornea,' p. 138. Basel, 1856.

the solution of the problem is to be found in the close parallel to be traced between arcus senilis and the other instances of fatty degeneration which have been described in this paper.

The pathology of fatty degeneration may be summed up in a few lines. And, as a preliminary, I must confess that a very attentive study of fatty degeneration continued through many years has much diminished, in my eyes, the importance of the specific character from which the process takes its name. Grave as are the diseases with which it is connected and on which it ensues, the fatty change itself is comparatively of little moment, and quite secondary to the degeneration, in date as well as in importance.

It is an infiltration of fatty matter which is derived immediately from the passing blood, or is made on the spot under its influence. And, as far as concerns the human subject, it is not the physiological form of fat, margarine, which is thus infiltrated, but oleine.

The structures which are infiltrated in this manner must be previously disorganised, the infiltration being a consequence, not a cause, of the disorganisation. Structures in healthy vital activity either do not admit of such infiltration; or, where their physiological constitution allows of this, they have the power of clearing themselves of the oil. They must be disorganised, but they must not be dead, as far as death implies liability to chemical decomposition or disconnection with the current of the circulation. For, experiments show that, under such circumstances, fatty degeneration, as expressed by the formal replacement of healthy structure by fatty matter, never ensues.

Article IV.—*Case of Popliteal Aneurism cured by Compression.* By Benjamin Barrow.

JAMES PERKINS, aged 31, a strong, robust man, a coal-heaver by occupation, was admitted into the Isle of Wight Infirmary on Wednesday, April 17, 1867, on account of what he termed 'a large painful swelling of the right knee.' The man gave the following short history: that 'he first observed the swelling a fortnight ago, that it had enlarged, and became more and more painful day by day.'

Examination discovered a large swelling extending from the

ham around the entire knee-joint; considerable pulsation was conveyed to the touch over the whole surface, and equally clear was the bruit de soufflet, heard with the stethoscope. The knee was bent at nearly a right angle. There was much pain, the skin was tense and hot, and so acute were all the symptoms that it became a question whether the femoral artery should not be immediately tied; but I preferred waiting, as pressure commanded the pulsation. Pressure, however, was not regularly commenced until 3 P.M. of April 19, for want of the proper instruments. On the first morning of the patient's admission the measurement around the knee was 16⅛ inches. In twenty-four hours this had increased to 17½ inches.

On the 19th, at 3 P.M., the first application of Carter's tourniquets was made—one to the groin, the other about one-third below Poupart's ligament, the pressure being kept up first by one, then by the other, and occasionally altogether remitted for a short period.

At 1.30 A.M. of April 20, the measurement was still 17½ in. and from this it grew slowly less until June 3 when it had diminished to 15⅛ in. The pulsation, bruit, and tension of the skin became day by day less apparent, the tumour at the same time becoming more and more consolidated.

On June 3 neither could pulsation be felt nor bruit heard; compression was, therefore, altogether omitted. After thirty-six hours we again perceived some evidence of the sac not being entirely obliterated. The tourniquets were reapplied and pressure kept up from time to time until June 17, being fifty-nine days from the commencement of treatment. From this date until the present time, August 24, every symptom of aneurism has disappeared, except the remaining hard tumour in the popliteal space, and which, from being as large as the largest orange, is now about the size of a pullet's egg. The knee has become gradually straight.

The man was a very restless subject, accustomed to partake rather largely of strong drinks: he was very sensitive to the least pain or inconvenience, and there is no doubt that frequently during the process of cure he slackened the tourniquets, especially at night, and to this cause may be attributed the length of time the cure occupied. He was discharged from the Infirmary on August 6. No particular medicinal treatment was necessary, and only a dose of opium was given occasionally. A generous diet was enjoined from his first admission. The swelling has now, August 1868, diminished to the size of a walnut.

ARTICLE V.—*On Rickets.* By SAMUEL GEE, M.D. Lond.

WHEN Whistler and Glisson appropriated the name of morbus puerilis to a special form of disease, they hardly exaggerated the importance of the position which rickets holds among the diseases of early life. In the present paper I purpose to make a few remarks upon some of those points in the history of the children's disease, which are still the subjects of dispute, or which are (so far as I can judge) not sufficiently known to the profession at large. It is not my intention to write a treatise upon rickets, nor indeed is it necessary, so peculiarly good are the works extant to which the reader may be referred; especially (i) A series of three lectures on Rickets, by Sir William Jenner, 'Med. Times and Gazette,' 1860; (ii) 'Clinique Médicale,' par A. Trousseau, 2me édit. vol. iii., 1865; (iii) 'Die Pathologie und Therapie der Rachitis,' von Dr. G. Ritter von Rittershain, Berlin, 1863. I shall comment upon (1) The frequency of rickets and the age at which it occurs; (2) Latent rickets; (3) Rickety deformities; (4) Dentition in rickets; (5) The apyrexia of rickets; (6) The urine; (7) Visceral disease; (8) The spasmodic disposition; (9) The nature of rickets; (10) The treatment; and, lastly, I shall make a few notes upon the bibliography of the disease.

1. *Age and Frequency.*—The following table is drawn up from patients who were examined by myself at the Hospital for Sick Children in the course of the year 1867:—

Degree of Rickets	1st Year				2nd Year				3rd Year		4th Year		Totals	
	Under 6 months		6 to 12 months		12 to 18 months		18 to 24 months							
	m.	f.	m.	f.	m.	f.	m.	f.	m.	f.	m.	f.	m.	f.
Slight . .	18	9	34	29	33	18	17	13	13	10	—	1	115	80
Considerable	—	2	34	18	45	27	24	22	15	25	5	3	123	97
Great . .	—	1	8	1	18	12	16	9	11	7	1	3	54	33
Extreme . .	—	—	—	1	—	1	—	—	1	—	—	1	1	3
Unspecified .	2	—	13	6	16	13	15	17	18	16	8	5	72	57
Rickets . .	32		144		183		133		116		27		365	270
													635	
All Diseases*	462		391		420		348		447		287		2,355	

* Including Rickets.

The ages given in this table are those at which the children first came under observation.

And so 30·3 per cent. of sick children under two years of age were rickety. This number agrees closely with that which Ritter gives as the result of his observations in the medical Poliklinik at Prague, viz. 31 per cent. of children under five years of age.

	Under 6 months	6 to 12 months	2nd Year	3rd Year	4th Year	5th Year	Totals
Male .	47	103	89	32	9	3	283
Female	44	72	65	30	6	4	221
Totals .	91	175	154	62	15	7	504

Out of 1,623 children seen. A comparison of the tables will show that it is highly probable that the proportion of rickets in children under two years of age was much greater in Ritter's practice than in mine; but, unfortunately, the data given by Ritter do not enable me to say positively that this was so. I can well believe that the frequency of rickets established by these tables may surprise some readers. Many years elapsed before physicians thought worthy of their notice a disease which the common people had discovered and named; but now-a-days there is no reason for belonging to a pre-Glissonite epoch. Nor is rickets peculiar to the poor; the severer forms may be so, but slight or even considerable rickets is rea'ly common in the children of the comparatively rich.

'Never congenital, rickets rarely occurs before the ninth month, scarcely ever after the second year.' (Boerhaave, 'Aphor.' 1481.) Never congenital: this, I believe, is not far from the truth; but the question of congenital rickets has never been fully investigated, and may be commended to those engaged in obstetric practice. A preparation put up by Lambl in the Prague Kinderspital Museum is the only instance of which there is a complete and uncontradictory account; of this case, details and a drawing can be seen in Ritter's book: the child was stillborn. In Mr. Stanley's work on 'Diseases of the Bones' (Lond. 1849) he alludes to a specimen of congenital rickets contained in our Hospital Museum. The preparation referred to is probably that which stands in the catalogue as A. 142. Now, in this skeleton I must say that I can see nothing which removes it from the category of that 'rhachitis congenita'

of which Rokitansky speaks ('Lehrb. der path. Anat.,' 3te Aufl. i. 43); a congenital dwarfishness of the skeleton attended by unspecified or unspecific deformities being the sole grounds for the name of rickets, but grounds, it seems to me, insufficient.

But the aphorism greatly underrates the frequency of early rickets. Unquestionable beading of the ribs may occasionally be found in infants of only three or four weeks old; at three or four months of age rickets is really common. It is best to take our grand criterion of the existence and degree of rickets from the state of the ribs: 'tabes pectorea' was a name imposed by Boot before Glisson invented his 'rachitis,' and certainly the pectoral signs of the disease are more easily discovered than any other, and when discovered more indisputable; they afford a clue which will lead us to the detection of rickets in its most obscure manifestations. Moreover—and this is the matter in question just now—I believe that no characteristic alteration in the bones (an essential element in the diagnosis of rickets) can be discovered more early than the beading of the ribs. It has been said, I know, that the cranio-tabes of Elsässer precedes the other osseous lesions by a month or two; true in exceptional cases this assertion may be, but that it is true as a general rule I doubt or even deny. It is best to trust to the ribs.

Rickets which begins to show itself late must be distinguished from rickets which takes on, late, a tendency to increase. Rickets supposed to be of late occurrence is mostly of the latter class; the children become slightly rickety under twelve months, yet they cut a few teeth and are able to stand or even to walk; then, some time in the second year, the rickets increases, and the children are taken off their feet. In ordinary practice it is obviously difficult or impossible to fix the time up to which any patient whom we may see was not rickety: for my own part, I certainly never recognised rickets setting in 'de novo' later than at twenty months,* and am therefore inclined altogether to agree with Alfred Vogel when he says that rickets never attacks a hitherto intact child after the first dentition;† nay, even to concur with Ritter (p. 54) that rickets does not often begin later than the first year of life.

2. *Latent Rickets.*—There are, apparently, all possible gradations between simple genuine debility and that special form

* Male, 20 months, has fourteen teeth; beading of ribs considerable; cut first tooth at six months; weaned and walked at twelve months; began to get weak in legs and loins six weeks ago; has sweated much for three weeks.

† 'Lehrb. der Kinderkr.' Erlangen, 1863.

of debility which we call rickets. So that sometimes the evidences of rickets are no more than in the following cases. Female, 12 months, just walks, sweats much, slight beading of ribs, no enlargement of ends of long bones, fontanelle an inch square, no teeth. Male, 2 years, ribs scarcely beaded, no enlargement of ends of long bones, can neither walk nor talk, sweats, fontanelle very large, five teeth uncut. Female, 3¾ years, can hardly stand, no enlargement of ends of bones, four teeth uncut. Examples, we might call these, of rachitis larvata, almost passing, as I have said, into the domain of genuine debility. Let us speak of a few special forms of incomplete or masked rickets.

i. Pseudoparaplegia, 'veluti inferiorum artuum paralysis' as Stoll says: the rickets falling, as it were, upon the muscles, the bones being left, in some cases, comparatively unaffected. An exaggeration, this, of the inability to stand or walk which is so constantly met with in rickets: that the muscles are at fault more than the bones is shown by those other cases in which, notwithstanding great softening of the bones, children are able to walk at twelve months of age, and become bandy-legged in consequence. We may lay down this rule. That a child who is not idiotic or weakened by some recent acute disease, and who cannot walk at eighteen months of age, is either rickety or paralysed. If the resolution of the legs does not soon improve under treatment, it is not rickety.

ii. The ligaments sometimes become much relaxed when the affection of the bones is not great.

iii. In partial rickets there is an analogous condition, the disease is limited to a certain set of bones: e.g. Female, 20 months, sixteen teeth; head of marked rickety shape, fontanelle large, sweats; ribs not at all beaded. Female, 2 years, great deformity of chest, yet none of head, fontanelle closed. One most important form of partial rickets is delayed dentition, which is, no doubt, in and by itself a proof of the presence of rickets. And the innumerable maladies attributed to delayed dentition are, for the most part, mere coincidences thereof, and, in fact, real consequences of the children's disease.

iv. I will here allude to the pains of rickets. We do hear such expressions as this, 'The child suffers great agony when he is lifted.' Sometimes periosteal in nature (as Vogel suggests), sometimes the result of emaciation (as Ritter will have it), these pains may be; but sometimes they are distinctly muscular: e.g. Male, 3 years, has all teeth, head rather square, wrists moderately large, ribs beaded to considerable degree, pretty fat,

cannot stand; when the legs are handled he cries with the pain, and when asked where he is hurt he points to the part touched; to press gently upon the muscles of the loins or belly causes an equal amount of pain; no complaint when pressure is made upon the bones; when told to put his legs down he does so slowly; told to lift up his legs he takes hold of them carefully with his hands, being apparently unable to raise them otherwise: in six weeks he was able to walk.

3. *Rickety Deformities.*—A. The Head. The skull undergoes changes in thickness, in size, and in shape.

i. Change in thickness, especially the cranio-tabes of Elsässer, to whose book ('Der weiche Hinterkopf,' Stuttgart, 1843), or to Alfred Vogel's Lehrbuch, the reader may be referred for full information upon the subject.

ii. Change in size—that is to say, an increase in size—has been spoken of by all writers upon rickets from Whistler downwards: 'morbus exhibet magnum caput,' says Boerhaave. But no doubt, as Ritter has shown, the enlargement, in the majority of cases, is relative only and not absolute; the growth of the skull is not retarded to the same degree as the growth of the face, trunk, and limbs; and for this reason the cranium does appear disproportionately large. But sometimes the cranium is absolutely enlarged, especially in cases of comitant hydrocephalus or hypertrophy of the brain: although certainly hydrocephalus is not uncommon in rickety heads which are not at all increased in size; and when they are increased in size it is by no means clear that the hydrocephalus is the cause and not the effect of the enlargement. But of this hereafter.

iii. Change in shape. This is of two kinds: 1 Increase in the length of the skull (dolichocephalus); 2. Diminution of the roundness. The longheadedness has been insisted upon by some authors as the especial rickety change. But, beyond all question, unnatural squareness and flatness of the cranium is a much more common condition: the skull tends to lose its natural arched shape, and to become flattened at top, in front, behind, at the sides; the frontal and parietal protuberances are increased: the frontal, coronal, sagittal, and sometimes even the lambdoid sutures are depressed. Partial exceptions to these characters are sometimes met with, but are not such as to modify the general truth of the description. For instance, the sutures may be obviously elevated and not depressed, so as to form flat ridges and not flat furrows; when this is the case with the frontal suture to any considerable extent, the forehead becomes pointed instead of flattened, the skull presenting other

unquestionable evidences of rickets. The flattening frequently affects one aspect only; the depression of the sutures is often limited to one or two of them; according to the tendency (previously discussed) which rickets exhibits towards partiality.

iv. The sutures. A condition which is intimately connected with the depression and elevation of the sutures is this—that in many rickety heads the sutures certainly feel as if they had opened. This is to be observed most frequently in the sagittal, not rarely in the coronal, and sometimes in the frontal and lambdoid sutures. Examinations post-mortem are greatly needed in order to discover what material (if there be any) it is which exists between the separated bones. Since my attention has been drawn to this condition, I have only once had an opportunity of examining a head of this kind, or rather a head which had been of this kind: viz. A child of four months had well-marked cranio-tabes and separation of the sagittal suture; ten weeks afterwards the sagittal suture was still open, though less than previously; in a few days the child died, but, post mortem, the bones were in contact, and even slightly overlapping. That the open sutures are occupied by tissue which is not osseous there cannot be a moment's doubt: and as little that the apparent dehiscence is not fallacious, and not due to thickening of the bones at a short distance from their edges, for the edges are almost always very thin and depressible, although distinct. The edges of the bones when they have not dehisced are often in a similar condition. The intervening tissue may possibly be osteoid, but, if so, with little or no calcification. The sutures rapidly come together or fill up as the general rickety state improves. And, lastly, the skulls, of which the sutures have dehisced, are often very obviously much smaller than is natural to the age of the children.

v. The hydrocephalus. That is a remarkable passage in Whistler's thesis: 'Cerebrum valde flaccidum et sero irriguum est; in ventriculos quoque ejus exundabat madoris affluentia.' On this point I have little to add to what I said in the last volume of the 'Reports.' The effusion has all the appearance of having taken place merely to occupy what would otherwise be a vacancy in the cranium. The brain itself is unaltered. It does seem as if in rickets the cranium is the only part of the body which goes on growing to anything like a natural degree; the rest of the body is certainly dwarfed, the brain included. If this be so, hydrocephalus must result, and is really, as said before, scarcely deserving of notice, except that at first sight it may seem to be allied to the grave incurable form of chronic

hydrocephalus commonly so called. I have never had the least difficulty in distinguishing the two conditions. The intracranial effusion of rickety hydrocephalus exerts no pressure, and does not itself tend to alter the size or shape of the skull; the peculiar dislocation of the orbital plates never occurs; very often there is no murmur in the fontanelle. The only treatment required for the cure of rickety hydrocephalus is to cure the rickets.

B. Thoracic deformities. From the deformities of the head and cranio-tabes, we come to the deformities of the chest—tabes pectorea. In studying this topic, we must distinguish three things —the beading, the lateral depressions, and the transverse depressions. We should also endeavour to distinguish the consequences of rickets from the consequences of concurrent catarrh. The whole subject is so very perplexed that I will not venture upon its full discussion, but will only set down a few detached aphorisms.

In great rickets the beading may be almost the only change, the depressions being none or next to none.

The amount of depression depends very much upon the amount of impediment to the entry of air into the lungs.

If we look at the chest of a child past all suspicion of rickets (say a child of seven years old) labouring under a paroxysm of hooping-cough, we observe that during the obstructed inspiration the ribs between the sternum and their angles become straightened, and even incurved at and near the costochondral joints. A transverse constriction of the thorax on a level with or a little below the sterno-xiphoid articulation is so common a consequence of catarrh in childhood that scarcely any person is wholly free from that deformity.

The depressions of the rickety chest are nothing more than the lateral and transverse depressions just spoken of carried to a degree which is not possible in a chest which is not rickety. i. First as to the lateral depressions. Sometimes the only change is an undue straightness between the nodules and the angles of the ribs. A sulcus, when present, mostly occurs farther back in rickety than in other chests; it is behind the row of beads, and tends to involve the bone more than the cartilage. Beginning to be seen just below the fold of the pectoralis major, or a little above the level of the nipple, the furrow goes on increasing in depth as we trace it downwards, until it reaches the transverse constriction. Sometimes the cartilages are as much depressed as the bone, even in extreme rickets, so that the nodules come to occupy the very bottom of the sulcus.

More rarely, what depression there is involves the cartilages chiefly, the groove being in front of the nodules. ii. Next as to the transverse constriction. This is on a level with the sterno-xiphoid articulation, or a little below this point; the cartilages of the seventh ribs are depressed, and also, to a less extent, of the sixth and eighth; hence, on either side, the furrow passes obliquely downwards, in front of the nodules of the lower ribs, or along them, or simply across them, until it joins the lateral furrow at an acute or a right angle: the abdominal organs are forced down, and the base of the thorax looks expanded. The maximum amount of depression is mostly where the lateral and transverse sulci meet; for instance, this is, in many cases, at the costal cartilages about the level of the eighth rib. Above the transverse constriction the thorax assumes the shape of emphysema: 'presque cylindrique ou comme globuleuse, bombée en avant' (Laennec, 'A. M.' i. 307); modified of course by the lateral depressions. The lateral depressions greatly diminish as the rickets diminishes; the transverse likewise, but not to the same extent as the lateral.

4. *Dentition.*—The teeth tend to decay (owing to deficient enamel), and to drop out undecayed. As an instance of the delay of dentition, I may allude to the case of a boy of three years who had cut only eight teeth; also to that of a girl, who at two years and two months had only the two central lower incisors. The second dentition seems to be delayed as well as the first: e.g. Female, 7 years, old rickets, did not walk till four years old, has no teeth except the two upper posterior milk molars, the rest have decayed. Male, 7½ years, old rickets, did not walk till four years old, as milk teeth drop out no more come in their stead, has no permanent teeth.

5. *Apyrexia.*—Rickets, per se, causes, I believe, no fever.

Female, 2½ years old. Whole family, including father and mother, rickety when young. Never walked; sixteen teeth, one decayed; great rickets of the chest; anterior fontanelle ¾-inch square; sweats somewhat about head; unquestionably tender. Temperature, taken in axilla morning and evening for three weeks, varied between 96° and 98·60° F.

Male, 2½ years old. Fourteen teeth; head rickety; thorax beaded, with lateral sulci; face flushes and head sweats profusely when child is asleep. Temperatures, taken five times daily for a week, varied between 96·5° and 99°, except on one occasion, when the temperature reached 100·2°, apparently in consequence of a diarrhœa.

6. *Urine.*—The difficulty in collecting the urine of very

young children is almost insuperable: I give the ensuing tables for what they are worth.

Male, 3 years; rickety, unable to walk.

	Water	Phosphoric acid (grammes)			
	Cub. cents	Total	Alkaline	Earthy	Proportion of earthy to alkaline
May 20 .	260	·565*	·507	·058	1 to 8·74
„ 21 .	246	·708*	·631	·077	1 to 8·2
„ 23 .	265	·769	·702	·084	1 to 8·3
„ 25 .	235	·705	·646	·059*	1 to 11
„ 31 .	310	·527	·471	·056*	1 to 8·45
June 1 .	266	·532	·479	·053*	1 to 9

* Those figures marked * were not the result of direct experiment. Neubauer's process employed.

Male, 2 years; rickety.

	Water	Phosphoric acid (grammes)			
	Cub. cents	Total	Alkaline	Earthy	Proportion
Sept. 3 .	137	·569	·493	·065	1 to 7·57
„ 4 .	105	·399	·336	·053	1 to 6·4
„ 6 .	114	·256	·217	·034	1 to 6·4

The proportion of earthy to alkaline phosphates in the adult is said to vary between 1 to 2½ and 1 to 7.

7. *Visceral Disease.*—Ever since the days of Whistler and Glisson the glandular organs have received much attention from those who have studied rickets (or pædosplanchnosteocace, as Whistler would call it); and those who have read Sir W. Jenner's Lectures will remember that he maintains that the viscera often undergo changes quite peculiar. Not to repeat Sir W. Jenner's opinions in this place, I will only point out how far, for my own part, I have been led to modify those opinions. 'Dolet quidem me,' as Morton says of Sydenham, 'in hac dissertatione ab ejus sententia discrepasse: sed, pace tanti viri, mihi etiam liceat liberam philosophiam ubique professo, pro modulo meo, saluti humani generis hoc pacto prospicere et consulere.'

There can be no doubt that the change which Sir W. Jenner calls 'albuminoid' is met with in rickets: no doubt that this change is quite different from the waxy, lardaceous, amyloid

degeneration. But I find myself obliged to doubt either the frequency or the fatality of albuminoid disease. If frequent, it is rarely fatal; if fatal, very infrequent. I have never seen it to perfection, except in children who have died from accidental catarrh. I have never seen either progressive emaciation or death which I could lay to the charge of albuminoid disease. When we can feel the lymphatic glands with ease in an emaciated child I doubt whether, in most cases, this be not a consequence rather than a cause of the emaciation; at any rate, many of such cases recover. Times without number have I felt spleens more or less enlarged in rickety children; comparatively few of these cases have died; and when it has come to a post-mortem examination, never but once was the spleen found albuminoid, and in that instance the child was quite fat, and died in consequence of pulmonary catarrh. In the majority of rickety children having an enlarged spleen who die, the naked eye characters of the diseased organ differ in no respect from those presented by the enlarged spleens of hereditary syphilis, the ague cachexia, and that cachexia which we must call essential, inasmuch as it is not related to any known cause. The diseased state of the spleen referred to is excellently described by Bright under the name of 'fleshy hardness with enlargement': the texture is confused, fibrous, fleshy, tough, dry, not at all specially translucent, the Malpighian bodies indistinguishable. I very much doubt whether the splenic tumour in these cases of rickets is more than accidental. I mean that the children suffer from essential cachexia (the name is unimportant), and no wonder that they become rickety if they are of a suitable age. I may add that we sometimes find the organs possess an 'albuminoid' character to a very considerable degree in children who are not rickety.

8. *Spasmodic Disposition.*—Upon this subject I must again refer the reader to the 'Reports' of last year. Whatever I have seen since that paper was written has only served to confirm the opinions there put forth. Not that they were altogether new; far from it; they have been held by physicians ancient and modern: 'morbi frequentes convulsivi, epilepsia per vices, symptomata rachitidis sunt,' is the sentence of Stoll ('Mb. Chron.,' vol. i. p. 20).

9. *Nature of Rickets.*—Rickets is a debility which is due to common causes, and which assumes a special form at a certain age, because it is only at that age that special organic processes are going on and can be debilitated. We may compare it with chlorosis, which also occurs at a certain age, and, moreover, in

one sex only. And a pathologist who sees in chlorosis no more than deficiency of iron in the blood may fairly consider rickets to be due to lack of earthy phosphates in the bones. But the saying of Baglivi will remain true, 'subtiliori physico subtilior est natura.'

10. *Treatment.*—This must be radical. And in cod liver oil we possess a pharmaceutical agent worthy of a place beside iron, Peruvian bark, and mercury. We ought to lose no time over the symptoms of rickets; slight catarrh, diarrhœa, paleness, a tendency to fits, these will all disappear under cod liver oil: give expectorants, purgatives, styptics, and the rickets will increase under our eyes; nay, occasionally, it will even develope 'de novo' while a child is being treated for coughs, deranged bowels, and other apparently simple disorders.

11. *Bibliography.*—A person who wished for information respecting the early literature of rickets would naturally turn to Van Swieten: nor would he be disappointed, for, in the fifth volume of the 'Commentaries,' the history of rickets is set forth with all Van Swieten's learning. More recent writers have placed implicit faith in the accuracy of the great restorer of the Vienna school, and have, in consequence, perpetuated a few mistakes. For an example I refer to Trousseau. It may, therefore, be not altogether useless to make a few comments upon the commentator.

The earliest treatise upon rickets, apparently not known to Van Swieten, is the thesis of Whistler: 'Disputatio medica inauguralis, de Morbo Puerili Anglorum, quem patrio idiomate indigenæ vocant The Rickets. Daniel Whistler, Anglosaxonicus orientalis. Lugd. Batav. 1645.' The title-page itself will correct two errors. First, Trousseau's mistake in calling Whistler a German; indeed, the Anglo-Saxon was president of our College of Physicians in 1683. Secondly, Van Swieten's mistake that rickets is a mere corruption of rachitis: whereas, even from Glisson himself, it is clear that the word rachitis was adopted chiefly on account of its similarity in sound to the homebred rickets. Whistler supposes rickets to be a derivative of a Dorsetshire verb, to rucket, which signifies to breathe laboriously.

The next description of rickets is that of Arnold Boot, in the twelfth chapter of his diminutive book: 'Observationes medicæ de Affectibus omissis. Londini, 1649.' Boot had practised in Ireland, and also in Paris, and at both these places had been familiar with rickets; contrary to the opinion of Van Swieten, who had, however, not seen Boot's work. Doubling of the

joints is spoken of by Boot as another vulgar English name for rickets.

In 1650 appeared the first edition of Glisson's book. Van Swieten calls this the second edition, a mistake which has led to curious argumentations. Copies of this edition are probably very rare, the only one of which I know is contained in the British Museum. The second edition appeared in 1660, and is more common. An English translation was published in 1651. I need not say that the name Rachitis dates from Glisson; and we can hardly be surprised that it was quickly accepted, if only to escape from the heirs of the fertile invention of Dr. Whistler, his 'pædocephalarthroncia,' 'pædosplanchnosteocace,' and other mighty inkhornisms. With reference to the assertion of Glisson that rickets first made its appearance in Dorsetshire, it is surely noteworthy that Glisson was born in that county, and was twenty-three years old in 1620, the date assigned by him to the invasion of rickets. I think that Whistler gives the real explanation of the supposed novelty of rickets at his day in a very few words, where he says, 'ex pluribus symptomatibus, divisim veteribus notis, complicatus hic morbus videtur; secundum syndromen vero signorum quæ conjunctim pathognomonicum constituant antiquis incognitus fuit.' In like manner, exophthalmic goitre and locomotor ataxia are diseases new to the nineteenth century, as rickets was to the seventeenth. We may say, with all respect, that Glisson might have spared his pains in seeking for the causes, which, supposed to be of recent origin, had brought about the new disease, among circumstances which had, curiously enough, been chosen five hundred years before by the good bishop Hildebert as the subjects of especial praise:

> Anglia, terra ferax, tibi pax diuturna quietem,
> Multiplicem luxum merx opulenta dedit.

Article VI.—*Surgical Cases, Devon and Exeter Hospital.* By Philip Chilwell Delagarde.

In presenting another collection of cases I am mindful that 'the history of a cancer is of small value until we know its end.' I therefore finish two cases, reported in the second and third volumes. To these memoranda I add an 'epithelioma,' which I had overlooked.

I describe a few more operations on the hand: not to exhaust the subject, for as the variety of damage may be infinite, so are the modes of repair; but to show what can be done, and how to do it, and to encourage thought on this, as on any other subject of plastic surgery, so that more may be done by others than I myself may dream of now, or shall, unless the occasion presents itself.

To miscellaneous cases I attach brief comments, explaining the motives which guided me.

Jane Thorn is dead, having survived the removal of a scirrhous breast (described vol. ii. pp. 185, 186), upwards of seven years. After my last report (vol. iii. p. 324) the thigh enlarged enormously, but did not ulcerate. During the last three months she occasionally discharged bloody mucus from the rectum and vagina—whether produced by cancerous ulcers, or by mere pressure, was not ascertained; but probably by the latter, as there was little pain, and no fœtor. The progress of this case was—December 1860, the operation; January 1865, neuralgia of anterior crural nerve; July 1865, osteo cancer of femur; February 1868, death from dropsy, as I learn from Dr. Drake.

Eliza Wright, she too is gone, having survived the amputation of her breast six years and four months (see vol. ii. pp. 186, 187, and my further report, vol. iii. p. 324). The course her disease took was—November 1861, the operation; September 1865, scirrhous gland above the clavicle; January 1867, looked well, felt well, but scirrhous glands reached the ear; August 1867, great hæmorrhage from an ulcer in the neck, with most offensive effluvium, which ceased with the bleeding; March 25, 1868, she died, as Mr. Bryden of Uffculme informs me, of two hæmorrhages, in almost immediate succession. There were large fungated and fissured ulcers, but very little fœtor.

I will here refer to my own remarks (vol. iii. pp. 329, 330), on secondary scirrhous knots. The lady died, some months after, of atrophy, withering up, and fading away. Of the crop of keloids infesting the cicatrix and integuments of the bosom, one had been dissected out by the late Mr. Rudall; and the rest shrank up, and never returned; but Dr. Thompson, of Bideford, informs me that, at her death, there were still several tubercles on the back (decreasing rather than increasing); and that she had anasarca, but not much dropsy of the chest. There had been intense neuralgia of the sciatic nerve. This I have so often observed accompanying fatal cancerous cachexy, that I cannot think it accidental.

I spoke of keloid tubercles in the 'Address in Surgery,' 1860 (see pp. 65, 66). Having related successful cases, I mentioned another, in which I had removed 'one such scirrhous knot. After several years I saw this patient again. There had been no return in the cicatrix, but there were glands, of a suspicious character, above the clavicle. The woman was in no pain; but her breathing was affected, and her health generally failing. I have heard nothing of her for some time, but I suppose she has died of hydrothorax.' That was no unreasonable supposition, but it was unfounded. Last winter she brought a daughter to our eye infirmary. She had become a stout, elderly woman. I could hardly believe it was herself, which amused her much. Her breathing was free and her health restored. This is very like Case XXIII. pp. 319, 320, in the last volume of these 'Reports.'

It is a subtile and intricate subject, which I would that others should investigate, rather than risk any inference myself.

I.—*Epithelial cancer of the neck.*

William Thompson, æt. 64 (a ship's carpenter, who had wintered with Sir Edward Parry in Regent's Inlet, and had gone with him over the ice from Spitzbergen), became my patient for epithelial cancer. He was in person to be noted: a very large and handsome old man, grand and serene.

The cancer was disc-shaped, four inches in diameter, and an inch thick. It covered the sterno-cleido-mastoideus, the mastoid process, and the angle of the jaw. It grew from a cylindrical base, only two inches across, which it consequently overlapped; but, as this base was eccentric, more on the sterno-cleido side. There was a foul, excavated ulcer in the centre of the disc, which bled, arterially, at times. The whole was movable, as though it were outside the fascia, except where a slip dipped into the parotid gland, between the mastoid process and the ramus of the jaw. That was its ominous feature.

I undertook this operation reluctantly; but when advised by such colleagues as Barnes and James, I could not hesitate. Observe, those eminent surgeons assumed the curability of epithelial cancer. They thought this operation right; they hoped, but were not sanguine. The old man himself saw that I did not like it, and, the night before, came quietly down to me in the library, and asked if I thought there was great danger of his dying under the operation. I told him that, with my friends to help, I could guarantee his life. He said, 'If, sir, there

were such danger, I do not think I should be right in having it done; but, if not, I think I ought.'

February 2, 1848.—I cut round the base of the tumour, and its formidable character was revealed. The sterno-cleido-mastoideus was degenerate; the root, or base, reached, and perhaps surrounded, the sheath of the carotid and jugular. Notwithstanding its free motion, nothing gave way, the knife was required everywhere. At length the greater part of the parotid was taken out, and all the tumour, except what lay behind the great vessels. Many arteries were tied. There was no paralysis of the face.* The wound granulated freely, healing until not larger than a shilling. What remained of the parotid then showed unequivocal disease; and he left these parts in a hopeless state.

This case and Case VII. (vol. iii. pp. 309, 310) show what dangers beset operations on the neck.

If, in paronychia thecalis, the first bone of a finger be not killed, I always try to save it; for although the flexor tendons may have sloughed out, yet the power is not altogether lost. As regards the little or the fore-finger this is plain; but the action of the lumbricales and palmar interossei, even on the middle and ring fingers, must not be forgotten.

* I made a note to this effect at the time, but can hardly persuade myself that the 'pes anserinus' escaped to the full extent it seems to imply. The integrity of the main divisions may be inferred—of the upper especially, for there was no lagophthalmos. All the intermediate branches were, I doubt not, destroyed; but granulation, or cicatrix, would compensate for loss of muscular support. (See Case XII., vol. iii. pp. 313, 314.)

Certain cases of lagophthalmos occur to me. I knew a gentleman who, whilst in the wilds of Australia, had a sloughing abscess of the right parotid, resulting in palsy of that side of the face. The unmeaningly staring eye was shocking.

A nice-looking girl—a servant spoilt by indulgence—had a tumour of the parotid. I advised her going into our hospital. She took offence, and said she would go home to Plymouth. I named surgeons there on whom she might rely. This gave still greater offence—she would choose her own doctor, wherever she was. After that I heard no more of her for years. One day I encountered the appalling gaze of an odd, though neatly-dressed woman, and wondered what she meant. She turned, and I recognised 'the parlour-maid.' As she had been impertinent and wilful, I affected not to penetrate the disguise of her disfigurement; but I afterwards learned that the tumour and facial nerve had both been extirpated.

Within the last five years I have seen three cases of lagophthalmos from injury of the facial nerve. One of these patients attributed the paralysis to a severe blow on the malar bone; but I found the scar of a deep wound, between the ramus and the mastoid, which he had forgotten.

The 'levator palpebræ' and the 'orbicularis oculi' are antagonistic, but they are supplied, the one by the third nerve, and the other by the portio dura of the seventh. If the latter be destroyed lagophthalmos ensues, the antagonism ceasing.

The difficulty is to get skin to cover the first phalanx, and to fill up the gap in front. The following method may seem whimsical and fantastic, but it succeeds: and I do not see how else the object could be attained. In raising a flap from the back of the finger, I make it substantial by paring it from the bone itself.

II.—*Amputation of the finger by swallow-tail flaps.*

John Farrant, of Clyst, St. Mary, æt. 46, had thecal paronychia of the fore-finger of the right hand. The flexors were sloughing out through the front of the proximal phalanx, but the bone appeared sound. This I desired to preserve.

October 22, 1863.—I cut across the front of the finger half an inch from the tip. I next cut along the back of the finger from the proximal to the distal end of the second phalanx, and then around the nail, until I reached the two ends of the transverse incision. Returning to the front I cut from the middle of the transverse incision to the proximal phalanx, and swept round the ulcer, which opened into the sheath. Having completed the outline I dissected off the skin, with the underlying tissues, as close to the bones as I could, and formed two long, and tolerably substantial flaps. The digital nerves and arteries were included. Then I finished by dislocating the second from the first phalanx. (See Plate I. figs. 1 & 2).

I united the two flaps posteriorly by sutures, turned them forwards, and filled up the hollow in front of the remaining phalanx. Pus welled up from the palm for several weeks (for the patient had been importunate, and induced me to operate sooner than I liked), but eventually he got a remarkably neat and useful stump.

I have repeated this operation successfully.

III.—*Arm and hand contracted, and webbed up to the axilla after a burn.*

Charles Bradbeare, of Kentisbeare, æt. 2½, had been severely burnt six months before. The cicatrices were complete, and all excitement had ceased. The hand was bent to the utmost. The elbow was acutely bent, and the arm and forearm webbed up to the axilla, into which the hand was tucked.

August 1, 1863.—I divided the cicatrix connecting the forearm to the arm. After a few days' poulticing, the arm was extended by a screw splint. Various dressings were applied, and, on December 9, the wound was healed, and the arm straight.

Soon after he was made an out-patient, with directions that 'the whole arm be swathed with a bandage; and that the splint be constantly applied, except for a few minutes every day, when the elbow is to be thoroughly bent and exercised.'

He was brought back to have the cicatrix, which tied the hand down to the wrist, divided.

July 8, 1864.—This was done, so as to take off all restraint. After a few days the hand was extended on a hand-splint. The elbow seemed well, but I would not trust to appearances; so I had the treatment steadily pursued, putting on the extending splint as before, its lower end resting on the hand-splint. By the end of 1865 the cure of both contractions was perfect; but I directed the splint still to be applied at night, for six months longer.

I saw him late in 1866; the cure was complete, even the scars were hardly noticeable.

It is sometimes more convenient to apply the splint to the back of the hand and arm.

IV. – *Finger connected with the palm by a cicatrix.*

Eldad Wollacott, of Hockworthy, æt. 6. A thick cicatrix, produced by a burn four years before, extended from the centre of the palm to the second phalanx of the middle finger. It drew the finger into the palm, and webbed the adjoining fingers.

June 8, 1868.—I made an incision on each side of the cicatrix, its whole length, and raised it, flapwise, from the sheath of the flexors down to the palmar fascia. The webs were divided; the digital arteries and nerves were preserved.

A digitated hand-splint was bound on the back of the forearm, and the fingers were looped up separately. The divided webs were kept asunder. Bread poultices were applied.

24.— Stimulating or astringent dressings, as required.

July 11.—The wound was quite healed, and the webs and contraction cured.

The splint is to be continued for twelve months longer. With proper care there is not the slightest risk that this contraction will ever recur; but the care is the condition.

In all these cases I cut down to the fascia, or other subjacent structure. I use bread poultices for the first few days, and, after the wounds are healed, I have the parts carefully bandaged (which smoothes the cicatrices in an extraordinary degree), and continue to apply splints over the bandage, as above directed.

V.—*Lacerated hand.*

Richard Henry Bowden, of Ashton, æt. 13. Had a severe laceration, commencing in the palm, below the little finger, and extending obliquely across the ring and middle fingers. The proximal joint of the little finger, the second joint of the ring finger, and the sheath of the flexors of the middle finger were torn open.

August 16, 1867.—The edges of the laceration in the palm were trimmed. The little finger was taken off; but a flap was made out of its back and side, and turned into the palm—filling up the gap. I then took a flap from the back of the ring finger, disarticulated the second and third phalanges, turned the flap forward, covered the bone, and filled up a laceration in front of the proximal phalanx. The flaps were secured by interrupted sutures. I did nothing to the middle finger, but wrapped up the hand in a bread poultice. The flaps exactly filled the spaces where the skin had been destroyed, making a curiously neat though mutilated hand.

VI.—*Crushed hand.*

Daniel Pym, of Topsham, employed in a paper mill, æt. 12, had his right hand crushed and lacerated by a machine. The end of the thumb was split up into five or six strips, and the bone exposed and broken. All the fingers were totally denuded of skin, and their distal phalanges broken. The skin was torn off from two-thirds of the back of the hand. On the palm it was uninjured, as far as the roots of the fingers; but it was scalloped there like the indenture of a parchment. He was brought to the hospital, immediately, from the Wear Mills.

January 30, 1868.—At 7 P.M. I dissected the skin remaining on the back of the hand, as far up as the proximal epiphyses of the metacarpus, divided the interosseous muscles, and cut the four bones. Then I raised the entire thickness of the palm from the metacarpal bones. The scalloped edge of hard cuticle was saved, that the points between the indents might take the sutures. The dissections, back and front, were then connected; and the fingers, with their metacarpal bones, except the epiphyses, detached. I left the thumb untouched. All the palmar nerves were preserved. I tied the radial artery, as it entered the palm, and several branches besides. Neither the ulnar artery nor its arch was exposed; and the digitals were all beating in the flap, but not bleeding.

The raw surfaces were left open to the air until next morning. The flap barely covered the wound—for I would not have it strained—but the denticles helped. The hand was wrapped up in bags of cold bread poultice, which were continued for a week. Stimulating dressings were next applied. The sutures and ligatures came away, and the flap was supported by a strip of plaster.

February 13.—Luxuriant granulations required zinc lotion.

March 18.—The wound was healed, and the cicatrix a mere line.

The flexor tendons adhere to the palm in front of the cicatrix,* and the boy puckers and dimples the skin, as one might with the palmaris brevis. He is a sharp lad, and will turn this to account. The remnant of the adductor brings the thumb across. The thumb itself is shapely, the tags of skin having fallen into their places.

I mention the thumb more particularly, because over care defeats its own purpose. If I remove a finger, I do not put the flaps together with the same exactness as if I reckoned on a thorough union by 'first intention.' Such success is too problematical. I make the flap long enough, and thick enough; secure it at one or two points; lay on a bread poultice, and leave the rest to nature. Had I brought the shreds of this thumb into closest apposition by stitches, or compressed them into shape, they would have sloughed, or ulcerated, but under the soothing protection of a poultice, they found their own places. Fifty years ago, when surgical dressing was the pride of the dresser, union by first intention more often failed than succeeded. Even now I see tidy rollers, if too tight, inducing mortification. I am convinced that a looser mode is safer, on the whole quicker, and in the end neater.

VII.—*Severe laceration of the hand and wrist.*

James Gatheridge, of Cullompton, æt. 58, a waggoner, was knocked down (March 30) by his waggon, the wheel lacerating his wrist and hand terribly. Besides lesser injuries, the skin covering the lower third of the radius, thence up the back of the metacarpal bone of the fore-finger, all across the thumb, almost up to the second joint, then nearly to the root of the middle finger, down the palm to the wrist, including the whole

* Some years since I began to engage the tendon of the tibialis anticus in the cicatrix of Chopart's amputation, thus antagonising the gastrocnemius. I sent up three such cases to Mr. Hancock, who spoke of them with approbation in his College Lectures. In the present instance I did not contemplate the adhesion.

ball of the thumb, was in tatters. The extensor tendons of the thumb were exposed, as were all the muscles of its metacarpal bone; and its first joint was entirely laid open behind. But the capsule and ligaments of the wrist, though bared, were untorn; no bone was broken; the radial artery was beating, under the extensor tendons, from the radius to the palm; and the fold of integument connecting the index with the front of the thumb was uninjured. The hand seemed too good to take off, and I hardly knew what to select for removal, so I waited; only supporting the hand on a splint, and applying bread poultices. A great deal of sloughing ensued.

After seven weeks there was a clean granulated sore. The long flexor had sloughed out; the metacarpo-phalangeal joint was destroyed, and the thumb hung by the soft parts; but it retained its proper sensibility, and the palmar artery and its fork amply supplied it with blood. Where so much had been destroyed, I desired to utilize such skin as remained.

May 16, 1868.—I cut across the front of the thumb, near its extremity. From this cross cut I made two perpendicular incisions, down to the open joint. Paring the skin from the front and sides of the phalanges, I detached the thumb. The flap measured exactly three superficial inches, and contained the nerves and arteries. I turned it back, and fastened it to the skin, on the palmar edge of the wound, with a couple of stitches. Knowing how difficult it is to make a crude wound adhere to granulations, I put no restraint or pressure upon it. Of course it contracted, and curled into just half its original size. I cared little about that, for the two surfaces did unite, and I reckoned that, as the sore healed, the flap would be dragged out again to at least its former dimension. In this I was totally disappointed, for it remains a firm curled-up heap of skin. It had never occurred to my mind that the substance, cementing the flap to the granulations, would really be cicatrix, and that, whether it surrounded the flap, or lay hidden beneath it, the contraction would be equal, area for area.

It helped, however, to fill up the wound, though not to the extent I meant. The sensation of a thumb is retained by the flap, and the man is well satisfied—saying that he shall be able to rest the handle of a spade against it. I call it a cleat, which he, coming from an inland town, does not comprehend. The metacarpal portion of the thumb is regaining a freer movement, although enveloped in cicatrix.

In the last volume of these 'Reports,' p. 323, I described a

mode of preventing flaps from retracting upon bones after amputation. I have used it for the thigh, arm, and fore-arm. It is peculiarly adapted to the last, of which I give a case. I now clinch the pin with cork instead of metal. It steadies the pin quite enough, and does not irritate like a shot.

VIII.—*Amputation of the fore-arm, and pinning the flaps.*

Richard Lake, of Clyst, St. Mary, æt. 13, a brick-maker, had his right hand crushed in a pugging mill. He was brought directly to the hospital.

May 27, 1868.—I at once amputated the fore-arm by flaps, sawing the bones just below the upper margin of the pronator quadratus. Four arteries were tied—the interosseous behind the ligament. The tissues under the skin were more bruised than I expected. The flaps were drawn gently forward, and retained by a German pin, passed perpendicularly through them, immediately in front of the interosseous ligament. The pin was clinched by a cork. The edges were brought together by wire sutures, but space was left on each side for the ligatures and sanious discharges to come through.

June 8.—The pin was removed.

26.—He was well: the ends of the bones being thoroughly protected by a dense pad of skin.

Injuries, requiring amputation at the shoulder-joint, involve a shock, which loss of blood might render fatal. I devise my operation accordingly, and illustrate it by my latest case. I do not affect any flap in particular. I rather prefer the deltoid—not cutting within an inch or more of the clavicle, or spine of the scapula, that its circulation may be derived from a larger area. Neither do I transfix, but I outline the flap—a course more sure, though less showy.

IX.—*Amputation at the shoulder-joint.*

George Brooks, of Huxham, æt. 55, a strong farm labourer, fell from a height, April 26, dislocating the left humerus under the clavicle. The whole arm was bruised and swollen.

April 29.—After unsuccessful attempts in the country, and in the hospital, its reduction was effected. The arm remained numb, livid, cold, and pulseless, but without any swelling over the axillary or brachial artery. In a few days the circulation was improved, and some sensibility returned; but there were foul, deep-seated abscesses, and the fingers and prominent points sphacelated.

May 10.—An eschar formed over the brachial artery, several inches below the axilla. Port wine, brandy, and strong ale had been given freely. The tension of the shoulder subsided, but mortification spread elsewhere.

May 16,—I amputated at the shoulder. The subclavian artery was well commanded. With a large scalpel I formed a deltoid flap, not following the precise outline of the muscle, but making it broader below. I raised this flap, opened the joint largely, cut the tendon of the biceps, thrust in the fingers of my left hand, brought up the thumb into the arm-pit, and held the nerves, vein, and artery tight, close to the glenoid cavity. Then I divided what remained of the capsule, carried the scalpel down close to the humerus, and cut the tendons of the pectoralis major, and latissimus dorsi. The head of the bone was drawn outwards, and with a small amputating knife I took off the arm.

Not a drop of blood escaped from the axillary artery, which was tied with its vein.* On removing my fingers the circumflex arteries bled.† They were instantly tied—not an ounce of blood being lost. Small branches bled, chiefly in the lower flap. Altogether the bleeding was less than four ounces. An abscess in the deltoid was opened, and scraped out.

I deem it most important that the operator should himself hold the axillary vessels. I have had them held by a most able assistant, but the act was not so consentaneous, and more blood was lost.

I am vexed that the brachial artery was not examined. The man was said to have had some previous illness, but I have never doubted that the 'reduction' produced the sphacelus. He continued in a precarious condition for a fortnight after; but care, and the appliances of a well-appointed hospital, brought him through.

* I never saw any harm from including a large vein in the same ligature with its artery. Indeed, in old patients, whose arteries are brittle, I tie in the vein and connective tissue purposely. I exclude nerves, but I have often seen them tied, inadvertently, without ill consequence. If tendon or fascia be tied the separation is tedious.

† As the deltoid was massive I only raised the layer next to the skin, so the circumflex arteries were not divided until the capsule was quite cut through; and, as they were given off below the point where I held the axillary, they did not bleed until I relaxed my hold. If in a slender or puny patient, I raise the whole deltoid in the flap; the circumflex arteries must be wounded before the joint is opened. Then, should the subclavian not be thoroughly commanded, I tie one or both before I proceed. This also applies to double flaps.

X.—*Cyst containing blood.*

James Baker, of Newton Saint Cyres, shoemaker, æt. 53, had a fibro-cystic tumour, containing blood, depending from the tuberosity of the ischium, and the inferior sacro-ischiatic ligament. It was egg-shaped, and twice the size of a large orange. He dated it from Crediton Fair, and it was surmised that a kick might have occasioned it.

January 8, 1853.—I divided the integuments, and dissected up to the tuberosity. I then found the tumour extensively connected with the lower ligament. I was apprehensive of the pudic artery, but I felt my way, worked carefully, and detached the cyst without serious hæmorrhage.

The man was thin, but still the wound was deep. There was a tendency to bleed, and I admitted air for some hours—as is my regular custom now. I stuffed it with lint, which came out when suppuration ensued. It soon got well.

I have removed several encysted tumours from the tuberosity of the ischium; but in this case only was the ligament implicated to so great an extent.

I have seen the pudic artery wounded several times—once, if not twice, in lithotomy—but always after it had reached the ramus of the ischium. It will sometimes bleed furiously in adults. I was called up at St. Bartholomew's in the winter of 1818, when the snow lay deep. The 'sister' herself, half dressed, ran over for me. She cried out, 'She will be dead, sir, make haste.' I knew the terrible import. The patient, a coarse big woman, a street-walker, had been stabbed, apparently with a penknife, without warning or provocation, by an utter scoundrel, who escaped. She hardly felt it, but saw the blood streaming on the snow. She shrieked for help, and was brought up, almost dead, from the Fleet Market to the hospital, by some watchmen. The gush had ceased, but I have never seen anything like her clothes; they were drenched through and through with blood. I forced in compresses, and bandaged them down tight on the pudic artery. It was hard to bring her round at the time, but she recovered in a few days. In the other cases compression succeeded.

But supposing the artery wounded nearer its origin, I think it still might be tied. I would cut round the margin of the gluteus maximus from the sacrum towards the trochanter, turn up quick, so as not to open the bursa, and divide its lower fibres freely. The sacro-ischiatic ligaments would be disclosed,

and the pudic might be secured between them. I do not see why a resolute operator might not go on to an ischiatic or gluteal artery—a most severe operation, certainly; but, rather than allow his patient to bleed to death, he might attempt it, if pressure failed, and nothing else were to be done. In a thin subject it might be practicable, and it unquestionably would be less formidable than Bell's operation, and less dangerous than tying the common iliac, or the internal.

XI.—*Hæmatocele.*

Isaac Gosling, of Branscombe, æt. 37, had had hydrocele for four years; but, three weeks before admission, he had received a smart blow. The scrotum did not transmit light, and the tumour had been taken for hernia.

April 15, 1862.—I tapped with a trochar. Serous fluid, much stained with blood, escaped, but the tunic was still half full of coagula. Great inflammation ensued, and the whole scrotum threatened to mortify. A week after I laid it open freely (which I had better have done at first), and removed a quantity of putrid clots. The testicle, and the cavity of the tunica vaginalis, were strangely coated with a dirty-looking, hirsute lymph—coarse and shaggy as a hall-mat.

The wound granulated and healed; but for more than a fortnight the man was in great danger from irritative fever. After that, obstinate diarrhœa came on; which, resisting usual remedies, was at last subdued by opiate enemata. He was cured on June 5.

XII.—*Hydrocele of several cysts, and thickened tunica vaginalis.*

Louis Palmer, of Broadclyst, æt. 58, had a hydrocele, elastic, smooth, and unusually pale. Malignant disease was suspected, and hence the exploration.

November 27, 1863.—I made a slight puncture. Clear, straw-coloured fluid escaped, and the swelling was reduced about a third. I repeated the puncture higher up, and reduced it again by another third. There was still fluid on the cord. The tunica vaginalis was excessively thick, but there was no evident disease of the testicle; so I desisted.

The man fell very ill, and required great care and support. A slough, as large as a half crown, formed on the scrotum. This was freely divided, and poultices brought away an im-

mense quantity of putrescent matter. The cavity slowly granulated and healed, and he got well; for the upper cyst disappeared, and the testicle was healthy.

I can only explain this intense inflammation by some of the fluid having escaped into the cellular tissue. I once saw this happen from an injection with port wine.* A milky cataract, which may have been harmless for years, will set up excessive irritation if punctured.

XIII.—*Hydrocele, and disease of testicle.*

G. A., of Dawlish, æt. 62. Had hydrocele on both sides, but there was also disease, assumed to be malignant, of the left testicle.

July 25, 1863.—The right side was tapped, and fluid, of the usual quality and quantity, was drawn off. Then the left side was tapped. Some days after I removed the left testicle. There was scarcely any constitutional disturbance.

He went out cured on August 26, the castration of the left side having radically cured the hydrocele on the right.

XIV.—*Hydrocele, recurrence after injections, and cure by seton.*

Not long after I began to use iodide injections for hydrocele, a fine vigorous fellow, a gamekeeper, came under my charge. The treatment seemed wholly to fail, his hydrocele, in a few days, being as large as before. I repeated it, and failed again. After a short interval I fell back on Pott's method by seton. That cured the hydrocele, but he left the hospital looking like a man recovering from severe illness.

Failing another time, I contemplated 'incision,' or the 'seton:' but paused, thinking I might have been hasty in the former case, and that I had not allowed sufficient time between the operations. I waited, therefore, and watched the patient, who got well without further interference.

This has been so often confirmed that, when other surgeons complain of similar ill success, I advise them to wait; and, as they do not tell me that my advice misleads, I presume it answers.

* See 'Address in Surgery,' 1860, p. 43.

XV.—*Hydrocele (with irritable testicle) returning after injection.—Cure.*

F. L., of Exeter, æt. 21, had had a hydrocele on the left side for a year. He had noticed a slight uneasiness of the testicle of late.

March 7, 1868.—I tapped the hydrocele; drew off about five ounces of fluid, just tinged with blood, and injected tincture of iodine ʒ ij., water ʒ iv. As very little pain ensued, he was directed to walk about.

9.—But little pain. The tumour was as large as before, but not inflamed. The contents evidently translucent.

11.—He was made an out-patient, and returned to his employment.

18.—The tumour was less.

23.—Still decreasing, the skin wrinkling, but the testicle slightly painful.

April 8.—The hydrocele was quite well; but a strumous testicle, of no great size, remained.

On the same morning, I operated on another young man, whose hydrocele returned. After waiting, to no purpose, I cured him by the seton.

Why certain diseases should attack certain organs, in preference to others, is sometimes hard to explain, although matter of common observation.

Neuroma is one of these. I find it in the female breast, causing severe neuralgic pains. I have removed such tumours when not larger than a millet seed, and, before dividing the skin, have secured them with spring-forceps, lest they should slip aside, and be lost in the fat. When a student, I saw one on the arm of a pretty cousin of mine, and advised her consulting Mr. Abernethy. She called on him, under the conduct of her governess, who would be spokeswoman. It was the raciest of interviews. But the most frequent seat is the calf of the leg. A few years ago, I went down to Exmouth to see an elderly gentleman, who, having lost his leg from an accident in the hunting field, had severe neuralgia in the stump. By friction he could make the neuroma quite perceptible—for it was erectile. It seemed to be the end of the nervus communicans poplitei. I told him I could easily remove it, but that he should first try aconite, externally. He sent me word, several months after, that he was much relieved, and content.

It does not confine itself to the branches of the popliteal, but, as far I see, affects nerves which either are, or are to become, cuticular. In hysterical women, I meet with neuralgia of the twig which curls on the inside of the knee, and I have known it there in men who hunt; but there has been no tumour in these cases. The wounds, after I have operated, have been slow to heal; but that may have been accidental.

XVI.—*Neuroma on the calf of the leg.*

Many years since I dissected a superficial neuroma, nearly an inch long, from the calf of the leg of a woman of 75. I cannot find the memorandum, which I certainly made. Erysipelas ensued, and she had a very tedious recovery. She was so ill that I regretted having operated on so old a patient. She left the hospital enfeebled, but cured.

XVII.—*Large neuroma on the back of the leg.*

M. A. Locke, of Mariansleigh, æt. 40, had a tumour the size of a pullet's egg, on the nervus communicans poplitei. There was dullness of sensation where the nerve was distributed. It was of two years' growth.

March 28, 1855.—I cut down on the tumour, and dissected it out, with a bit of the nerve at each end. Erysipelas ensued. The wound was not healed until May 17.

XVIII.—*Neuroma of the peroneal nerve.*

Martha Madge, of Crediton, æt. 33, had had a painful tumour, close to the fibula, for several years. It was a neuroma of the peroneal nerve, while deeply lodged between the peronei muscles. It was the size and shape of a blackbird's egg, but appeared larger and flatter through the fascia.

November 27, 1861.—I divided the skin and fascia, from above downwards, and dissected it out of the muscle, with half an inch of nerve at each end. An artery, accompanying the nerve, was tied. The wound was slow in healing; so I let her get up, and walk about, which was of service. She did not complain of insensibility on the outside of the foot, seeming too obtuse to distinguish the difference.

I have often remarked that wounds on the outside of the foot are very painful—perhaps from the distribution of this

nerve on the skin. I have twice seen tetanus after laceration of the outer ankle. Both patients died. I gave them quinine in large doses. One, an old man, did not derive the slightest relief from this treatment—the only case, within my own experience, in which it has so totally failed.

Adipose tumours are seldom remarkable, except for size. Now and then they occur, unexpectedly, in odd places. I was consulted by a farmer's wife for a large swelling under her tongue, which was thought to be a ranula. It did not feel or look like a ranula; but sometimes these tumours have very thick cysts. I cut in bravely, when out peered a nodule of fat. I traced it beyond the root of the tongue, and separated, with my finger, a tumour full the size of a hen's egg. Fortunately there was little bleeding, for I had only one pupil with me, and had made no preparation, beyond a dossil of lint and some tincture of muriate of iron.

XIX.—*Adipose tumour under the obliquus externus abdominis.*

John Middleweek, of Hittisleigh, æt. 31, had an adipose tumour of fourteen years' growth. It occupied the flank between the lower rib, the lumbar fascia, and three-fourths of the crest of the ilium.

March 4, 1848.—I divided the integuments, and a dense superficial fascia. Reflecting the latter, a curious appearance presented itself, glossy, yellow nodules cropping up in all directions, between the red, semitransparent fibres of the muscle, as if through a net. I cut these meshes, after which the tumour was readily detached from the subjacent parts. He was well in a few weeks.

He had been rendered dreamy by ether, and, being a local preacher, held forth with unction, but was unconscious of pain. He remained excitable for several days. I did not know what had become of him, but he called upon me last summer; just, I fancy, to renew our acquaintance. He was in good health, disposed to expound, and none the worse for having had his obliquus externus cut across.

I suppose fibrous tumours are liable to fatty, as they are to cancerous degeneration. I meet with equivocal growths, neither fibrous nor adipose exactly.

XX.—*Fibro-adipose tumour on the back.*

Edwin Ford, of Exeter, æt. 73, formerly a farmer, who had lived freely, and was florid, good looking, and gouty, had a large prominent tumour extending, by the side of the spine, from the scapula to the ilium.

June 7, 1865.—On dividing the skin it looked like a huge hernial sac full of intestines. It turned out to be a fibrous tumour passing into adipose. I saved all the skin, which was not too much in the end. Several small arteries were tied. The tumour weighed 2 lbs. 7 oz.

There was much sloughing in the cavity, and for eight days he had unceasing hiccough. The sloughs gradually separated, and the hollow granulated. He was in great danger for a fortnight. He got well in eighty days, and continues in good health.

XXI.—*Subfascial fibro-adipose tumour of the knee.*

Thomas West, of Topsham, æt. 56, had a tumour, the size of a cricket ball, on the outside of the thigh, beneath the fascia, in the hollow formed by the condyle, the vastus externus, and outer ham-string. It was of two years' growth, was increasing in size, and was excessively inconvenient when he walked. It was so firm and elastic that, having been mistaken for a bursa, it was pierced subcutaneously. There was no fluid, and it was then considered fibrous; which it really was, but degenerating into adipose. I divided the skin and fascia, and dissected out the tumour. A section showed its true character—fibrous substance with fat intervening. The wound was sound in three weeks.

XXII.—*Fibro-cystic tumour on the patella.*

I have no other note of this remarkable case than the following, which Mr. James inserted in the catalogue of our hospital museum.

'703. A cyst over the patella. The bursa was beneath. The contents resembled mashed malt. Woman æt. 46. Admitted September 26. Operated on three weeks after. Went out perfectly well two months after. It weighed 18½ ozs. Mr. Delagarde has the cast. This case might well be confounded with enlarged bursæ.' (See Plate I. fig. 3.)

The tumour looked as though it extended beyond the patella; but I was satisfied it did not, and so it proved. I made

a perpendicular incision, and reflected the skin, of which there was not too much. It was put together with twisted sutures. The bursa on the patella had not been opened. After getting well, there was no tendency in the cicatrix to split open.

XXIII.—*Fibro-cystic tumour on the front of the knee.*

Elizabeth Gibbons, of Tiverton, æt. 40, had a cyst covering the patella, and, unlike the previous case, adherent beyond its margin. The contiguous surfaces of the cyst and bursa were consolidated.

February 18, 1865, I made a longitudinal incision, and reflected the skin. The tumour was then carefully separated from the investments of the joint. The bursa on the patella was opened, and in part removed with the cyst. The skin was put together with twisted sutures. It healed in forty days; but after that she had troublesome cellulitis. She was a bustling, lively, reckless woman, who would not believe that anything could hurt her, and did not much care if it did. I saw her a year after the operation, when she was still in an unsatisfactory state.

February 5, 1868.—I saw her at Tiverton. She was lame, but otherwise well.

The tumour might originally have had no attachment to the bursa. In other respects these two cases were much alike.

XXIV.—*Loose cartilage in the knee.*

John Underhill, of Alphington, æt. 26, had felt a loose cartilage for two years. It was so large as to occupy one half of the joint, yet admitted of some movement. Its outer side was a fourth of an inch thick; it got thinner in the middle, thickened a little again, and gradually lost its pearly character, until, inwardly, its margin looked semigelatinous.

March 3, 1849.—The skin, well drawn up on the outer and lower side of the patella, was divided, the joint opened, and the cartilage drawn out. It covered the whole of the outer glenoid cavity. The cut into the capsule was more than an inch long, and the cartilage curled completely upon itself in coming out. It was moulded between the glenoid cavity of the tibia and the condyle of the femur.

The edges of the incision were drawn together by strips of sticking plaster, and protected by lint steeped in blood. The dressings were removed on the 21st, when the wound was sound.

XXV.—*Floating cartilage of the knee with attachments.*

William Wright, of Morchard Bishop, æt. 20, a robust farm labourer, had a movable cartilage in the left knee. It lay between the inner condyle, the head of the tibia, and the ligamentum patellæ. It rolled under pressure, but did not slip away. It seemed the size and shape of an almond, but was not well defined. This obscurity made me depart from my usual method of simply drawing the skin aside and cutting into the joint.

December 17, 1866.—I drew up the skin, keeping the cartilage against the tibia with the fingers of my left hand. I made a semicircular sweep of three-fourths of an inch radius, continued it half an inch up on each side, and outlined a flap like a stilted arch. I raised this flap, exposing the capsule, which was thickened, congested, and inelastic. I divided the capsule, for an inch, along the loose cartilage. It did not start, although I was pressing from above; so I nicked upwards (see Plate I. fig. 4). The middle bulging out, I seized the body with bull-dog forceps, and found its two ends fixed to the margin of the glenoid cavity. Mr. Cumming, who assisted me, drew it forward or backward, as required. Carefully excluding air, I detached the two ends with a very slender bistoury. One or two drops of synovia escaped when the joint was opened, but none afterwards. The flap, when laid down, effectually covered the opening. It was secured by two sutures, and a pledget of lint steeped in tinct. benzoini comp., which is not so trustworthy as blood. The limb was then laid in a McIntyre's splint.

The cartilage was one inch and a half long, and one-third where thickest. It was fusiform. The hinder end adhered by a fine point; the anterior, after becoming nearly as small, enlarged abruptly, like a head set upon a narrow neck. This head was a flat disc, like the tail of a leech. Indeed, except that it was rounded instead of flat, the body was like a leech, but of a bright pearl colour.

He was quite easy for two days; but, on the third, he had great pain, with erythema, down the saphena vein. This was preceded by a rigor and an exceedingly hurried circulation. The pain subsided in a few days under soothing fomentations; but, on January 8, a large abscess formed on the inside of the mid-leg; and on January 17 two more appeared, one over the tibialis anticus, the other over the internal ham-string. These

abscesses were beneath the fascia, and were opened, with great constitutional relief. As the joint was unaffected, I presume the opening into its capsule had healed at once.

February 5, 1867.—He had deep-seated pain under the rectus femoris, as though the shaft of the bone were inflamed. There was little heat or tenderness of surface; but the whole thigh swelled, and there was fluid under the muscle.

7.—The pain and swelling decreased.

12.—The pain and heat returning, I opened the abscess at the outer edge of the rectus—which had been floated up. More than a pint of pus was discharged.

March 3.—I passed a draining tube into the cavity, right under the rectus.

6.—The two abscesses below the knee had healed.

8.—The abscess on the inner condyle, which had partially discharged itself under the flap, still retaining pus, I passed a draining tube into it.

19.—Except that the knee was stiff the limb was restored, and he was made an out-patient.

Some time after he wrenched the knee, which enlarged the motion; but such irritation was set up that I again took him into the house until it subsided.

November 20.—He was well again. I still fear to bend the knee forcibly.

All the abscesses were preceded by severe rigors, and for some weeks there was intense constitutional derangement. Although suppuration was confined to one limb I treated it as pyæmia. I have, however, slight experience of that formidable disorder, and write under correction.

In Vol. ii. p. 185 of these 'Reports' is a note and plate illustrating my mode of sawing the bones in 'Resection of the knee.' I now give two cases in which the osseous union was long deferred. Both are well represented in that plate. I have never yet failed in obtaining a well-shaped and useful limb by resection—which very nearly represents the result of the large experience of our hospital.

XXVI.—*Resection of the knee: oblique sections.*

Sumuel Mogford, of Tiverton, æt. 23, a clock maker, had ulcerated cartilages of the knee-joint. The patella was united to the femur. For some months the limb had been carefully supported on a splint, to favour anchylosis. At length his health sank dangerously.

July 5, 1860.—I made an H incision, broke off the patella from the femur, and sawed the condyles and glenoid surfaces obliquely. The limb (in a McIntyre splint) was swung under a common cradle suspender,* and trimmed by guys of buckled tape. I never use the long interrupted splint.

When he left the hospital there was ligamentous but not osseous union—so he wore whilst away from us an instrument which kept it firm and straight. He returned two years after. The femur and tibia had been firmly united for a twelvemonth; and the limb, shortened little more than two inches, was useful and of excellent form.

This was the first case in which I made the oblique sections and the last in which I made the H incision. I have since taken a single flap from the front. I always remove the patella.

XXVII.—*Resection of the left knee—The right hip dislocated.*

William Blake, æt. 17, a scrofulous youth of squalid complexion, whose right thigh had been dislocated long before by disease, was sinking under abscesses and ulcerated cartilages of the left knee.

February 1, 1861.—I raised a flap from the front of the knee, took out the patella, and sawed off the entire epiphyses of the femur and tibia to make the two limbs of a length. The sections were oblique as in the preceding case. For a time all went on favourably, when inflammation recommenced in the right hip. Abscesses followed, and the union of the resected bones was suspended. The outline of the limb was, however, undisturbed, and sufficient consolidation was at last effected to justify his removal. The weather was hot, and country air afforded his only chance. I heard nothing of him until nearly three years after, when I fell in with a woman, his neighbour, who told me he had recovered, and was able to walk as before, with a crutch. I have assumed osseous coalescence, but possibly the bones may not actually have grown together. Their broad surfaces may only be connected by fibrous tissue, and be held firmly in contact by bands of ligament, but the account I received implied a thorough reparation.†

As diseases of the bones approach the larger joints, they

* Mr. Henry Greenway, of Plymouth, has invented a suspender, the movements of which display great mechanical ingenuity. It has just been introduced into the D. & E. Hospital.

† The gaping wound of resection is reduced to the condition of a compound fracture by removing the articular surfaces; but a desperate injury of the knee is

become more difficult of surgical treatment. I give two cases of exostosis of the knee and shoulder, which were in dangerous proximity to the joints. I saw Mr. James take off an exostosis from the femur. His case, which did well, exactly corresponded with mine, except that the exostosis was smaller, which certainly increased, rather than diminished, the difficulty of the operation.

The neck of the humerus is very liable to morbid growths. This exostosis was not malignant, but it throws light upon the mode in which a hard protruding spike may be formed by the wearing away of a semicartilaginous tuber.

XXVIII.—*Exostosis of femur.*

W. R., of Exmouth, æt. 16, a remarkably fine lad, had an exostosis of the femur. It began at the inner ridge of the linea aspera, close to the knee, by a long and hard spike of bone which grew obliquely, upwards and inwards, under the vastus internus, and terminated in a tuberous osseo-cartilaginous head. The spike was three inches long, and the head the size of a small orange; but, when covered by the skin and muscle, it looked much larger than it really was. The exostosis was enclosed in an adventitious capsule.

March 13, 1866.—To uncover the tumour I divided the skin, the fibres of the vastus internus, and its large muscular artery. The latter was tied immediately. Then the spike was cut through, as near the knee-joint as I dared, with bone forceps, and the base was nipped smooth with gouge forceps.

The wound healed readily. Some time after I thought the remnant of the base, near the knee, prominent; but I believe it had merely become more conspicuous from absorption of the fat.

not absolutely hopeless. I made no note of this case, which must have occurred five-and-twenty years ago; but my recollection is clear.

A gardener of Mr. Divett, whilst trimming a hedge at Bystock, struck a pruning-hook into his knee. It entered on one side and came out at the other, dividing the ligamentum patellæ, and laying open the joint as if for resection. The cartilages were exposed, the capsule being opened nearly half-way round. I took out clots of blood from between the crucial ligaments with my fingers. The wound was dressed with adhesive plaster, and covered with lint steeped in blood. The limb was placed on a splint, something like McIntyre's.

The dressings were not touched for many days—I believe a fortnight—when the union was general. Great care was taken, for months, to prevent the leg being bent. He recovered perfectly, and walked as well as ever.

He was a fine, handsome young fellow. His wife came with him to the hospital, and brought the baby. She was as good looking as her husband. The group inspired my attempt to save his leg; which I confess was rash, and, though successful, hardly to be justified.

December 17.—It was in the same state, and he was discharged cured. He remains quite well, July 20, 1868.

XXIX.—*Exostosis of humerus.*

Francis Shears, of East Ogwell, æt. 24, had a composite exostosis of the humerus. It began eight years before. Its base, which was long, narrow, and very hard, extended from the neck of the humerus to the insertion of the deltoid. Its tuberous head projected between the outer part of the triceps and the deltoid. It was osseo-cartilaginous, and the size of a small orange. A spike of bone, a process from the same base, had pierced through the substance of the deltoid, and stood out an inch and a half through the skin—sharp, hard, and soiled from exposure.

September 6, 1862.—I divided the skin along the outer edge of the deltoid, exposed the noduled head, which was enclosed in a capsule, and cut it off with bone forceps. Then I cut the fasciculus of the deltoid, and uncovered the spike, which was as thick as one's thumb. I cut it off with a very strong pair of bone forceps. Then I nipped away the whole of the base with gouge forceps, leaving an oval aperture into the medullary cancelli two inches long and more than half an inch wide. It reached close up to the greater tuberosity of the humerus.

The skin was brought together by several silver wire sutures, and covered with a bread poultice. The arm was laid on a pillow, away from the trunk, to facilitate the union of the divided fasciculus of the deltoid. Scarce any disturbance of health followed, and he got well, as he remains, in little more than a month.

A far simpler operation is required to abstract a sequestrum from the outer than from the inner aspect of the thigh. I give a case of each.

XXX.—*Necrosis of femur.*

Isaac Gloyne, of Shebbear, æt. 24, had necrosis of the lower half of the shaft of the right femur. The new case of bone was more than an inch thick and excessively hard.

August 30, 1860.—I divided the skin and vastus externus, parallel to the tendon of the biceps, from the knee seven inches upward. Two dies were taken out of the case with a large trephine, and the bone between was cut away with pliers, chisel, and bone forceps. A sequestrum was found eight inches

long. It was cut through, the two pieces removed, and the cavity cleared.

The knee, which had been a little bent, became straighter; but the wound was not quite healed when he went out. He came again a year after, the surgeon of the village, a well-informed practitioner, assuring me that some dead bone was still there. I felt bone with a probe, but it did not move, and I very much doubted its being dead. However, as I was urged by the patient, and his friends, and his doctor, I opened the medullary cavity for about six inches. This was easily done, for it was closed up, not by bone, but by dense fibro-ligamentous membrane. It was the edge which had been felt, the cavity was empty. A small fistula remained when I last saw him, but he has a good serviceable limb.

Whilst he was under my charge for this necrosis, it was discovered that one of his fingers was contracted. I divided the flexor perforans in its sheath, using a curved tenotomy knife that I might avoid the digital nerves and arteries. The hand was extended on a splint, and the finger cured.

XXXI.—*Necrosis of the femur— Contracted legs.*

Sarah Shepherd, of Sidmouth, æt. 23, had necrosis of the left femur, resulting from a fever twelve years before. One large sequestrum, and several small ones, had exfoliated from the lower and outer part of the thigh. There was a cicatrix, ten inches long, from the outer condyle, following the linea aspera. The skin adhered tightly to the bone. Other scars indicated former abscesses as high as the trochanter, but no fragments had come through them. There was an open sinus inside the thigh, three inches above the knee, just in front of the sartorius. Through this a probe could be passed into the popliteal space, and dead bone distinctly felt. The knee, which was so bent that the heel touched the buttock, could not be drawn out, yet it was not anchylosed. The thigh was so everted that the head of the bone was prominent in the groin.

The right knee, bent to an acute angle, was stiff, but not anchylosed. Her appearance was most ungainly. The face indeed was pleasant and good looking, but the body was as squabby as a Chinese Joss, and with her legs tucked under her she looked like a huge child playing at 'hunt the slipper.'

As the soft parts were so agglutinated on the outside of the left thigh, and as the sequestrum could only be felt from the inside, I decided to get at it through the vastus internus, in the

interval between the saphena vein, the saphenous nerve, the femoral artery, and the capsule of the knee.

October 3, 1866.—I cut down to the bone from the sartorius to the condyle. The muscular artery of the vastus internus was tied. Then I divided the skin across, as far forward as the rectus—parting the fibres of the vastus internus, which were pale and indistinct. I applied a trephine to the femur, close to the linea aspera, took out a large die of new bone, and enlarged the opening with bone forceps. I seized the sequestrum with the lion-forceps, and cautiously, and with apprehension, drew it out. Nothing was hurt. The sequestrum was a thin jagged flake, in size and shape like the under shell of an oyster. It was the popliteal face of the femur, and must, when she was twelve years old, have occupied the whole space above the condyles. The muscles were much degenerated, the bulk of the limb consisting of hardened fat.

The wound was covered with a bread poultice. A copious and fœtid discharge ensued, which lasted, more or less, for months. In ten days, healthy granulation being established, a well-padded fillet was attached to the ankle, with a weight at the other end. The weight, hanging over the foot of the bed, drew on the knee, unbending it. It set up fever and inflammation, and I desisted; but after some few days I tried again. In a few weeks the knee made a right angle. It yielded slowly, and an extending splint was put on. The weight was continued, sometimes over the foot of the bed, sometimes over the side. At length the knee was so straightened that she began to exercise between parallel bars.

I had also been suspending a weight from the right foot, but, although that knee was the less distorted, it was the more intractable of the two. An extending splint was therefore added.

Both legs, acted on day and night by splints and weights, became straighter; but I found that, if I persisted, I should dislocate the right tibia backwards. I therefore

March 23, 1867, divided the ham-strings. I drew the skin well aside, cut down to the tendon of the biceps, passed in a Fergusson's lithotomy knife, outside the peroneal nerve, and divided the tendon subcutaneously. I did the same to the inner tendons, dividing the fascia extensively with the same knife. She soon resumed the splint and parallel bars, making great progress.

I thought a change would refresh her; so I sent her to Sidmouth for a month. After her return (the former treatment

being continued) I had her thigh frequently rolled inward. This exercise gave freedom of motion, and when she began to walk with crutches, I made her an out-patient.

I saw her on April 15, 1868. The right leg was restored—except that the ankle turned aside, as in a rickety child. The knee was straight, and gaining strength. She walked with one crutch.

The wound in the left thigh was healed. The knee was nearly straight, but the heel did not rest on the ground, and the limb still turned out. She looked remarkably well, had got rid of her loose fat, and gave remote promise of a nice little figure.

August 14, 1868.—I have seen her again, and begin to fear that the left hip will not admit of further correction. I intend setting her right ankle straight by the common apparatus.

XXXII.—*Leg distorted by a compound fracture.*

James Quick, æt. 29, a fine healthy man, a butcher, sustained a bad compound fracture of the left leg on March 18, 1866.

He had been in a local infirmary until he came to the hospital May 30, 1867. The leg (measuring along the spine of the tibia) was shortened an inch and a half. It was excessively distorted outwards, the ankle being full five inches out of line. The upper fragment of the tibia reached four inches below the knee. The lower fragment was displaced upward and backward; and, as I afterwards ascertained, a detached fragment was consolidated in. The whole was surmounted by an immense hemispherical mound of callus which stretched the integument to bursting. There was a large ulcer on this callus.

I directed the jointed stem used for a knock-knee, which in a few months reduced the bend by one-half. The ulcer was sometimes better, sometimes worse. After a while I let him move about, and twice sent him home for change.

A probe went three inches right down into the fracture. There I felt rough bone, but was not sure that it was loose. I assumed it to be a sequestrum, and, as the tense skin forbade all hope of healing the ulcer, I determined to open the callus, reduce its bulk, take out the dead bone, if any, and probably facilitate the straightening of the leg.

April 29, 1868.—I made a large crucial incision, dissected back the flaps, scraped off the granulations, and exposed the

callus. I trephined its face, taking out three large dies. The crust was an inch thick. I cut away the intervening callus with bone and gouge forceps, sawed out another piece, and made a rhomboidal aperture of about three inches a side. I feared to take away more, lest I might break the bone; but I had greatly reduced the prominence. I found only one sequestrum, the size of a sixpence. Thus I laid open the original fracture with its upper, middle, and lower fragments—now united.

He was generously fed and had 8 oz. of port wine daily. In a fortnight I desired him to lie on his left side whenever he could, with the foot raised by a pillow to keep his leg hollow from the mattress, and a sand-bag across it, below the knee, to press the limb straight.

May 15.—Exfoliations presented, where the trephine had worked. The granulations were profuse.

21.—The wine was given up, but he still took a pint and a half of strong ale daily. He left the 'separation' for a 'common' ward. The leg was straighter, and the exfoliations had been cast off. He put on his old instrument for a day, but it had reached the limit of its action. His foot was therefore turned up, and a sand-bag attached to the knee was hung over the left side of the bed, and another, drawing upon the ankle, over the right side.

June 17.—The leg was almost straight. To lengthen it a sand-bag over the foot of the bed was added.*

* The beneficial effect of traction in diseases of the knee and hip is now fully recognised. Its value in broken thighs was known long since, but was overlooked, until forgotten. Mr. Erischen ascribes it to Dr. Buck, of New York ('Lancet,' vol. i. 1868). In the 'Address in Surgery,' 1860, I claimed to have been its inventor half a century ago. In speaking of fractures of the femur, I said, 'When I became House Surgeon of St. Bartholomew's, in 1818, I asked Mr. Vincent if he would allow me to treat his patients on a new plan, which I explained to him. He admitted that nothing could be well worse than the results of short splints and the bent position, and that I might try. I had read a great deal of French surgery before I went to London, and was much struck by the neatness with which they treated their fractures. Not but that it was much too nice to be applicable generally: so I set about reducing the methods of Desault and Boyer to the level of vulgar utility, for I have never used, if I could help it, any apparatus in a hospital which could not be had and used everywhere else. I took a well-padded splint, which reached from the foot to half-way between the trochanter and the crest of the ilium—not higher than the crest, for that is a mistake, any authority to the contrary notwithstanding. This I buckled round the pelvis with a broad tape, quilted with a narrow pad, or cat's tail. Another well-padded splint reached from the pelvis to the calf of the leg. Sometimes, if the limb was large, I put on a third narrow splint above. If the bone was broken in its upper third, I laid the limb on an inclined plane, as in the case of fractured patella. All this was, at that time, regarded as a novelty, and encountered some opposition, and more criticism. But another unheard-of appliance nearly raised a mutiny amongst the

19.—I ordered syr. ferri phosph. c̄ ʒj. thrice a day. The wound had nearly filled up, and was healing fast.

Aug. 10.—The leg firm and straight, but shortened more than an inch.

Necrosis of the tibia is so common that apology is due for introducing the following cases. It is essential, however, that the new bone should be strong enough to replace the old ere the sequestrum be removed; and I have selected three, out of many, to illustrate this principle.

XXXIII.—*Removal of necrosed tibia delayed for want of a new bone.*

John Dodge, of Chagford, æt. 12, a scrofulous and weakly boy, had necrosis of the tibia, ensuing upon recent periostitis. The shaft had separated from the epiphyses, new bone had not formed, and the leg was only partially supported by the fibula. I had splints applied, gave him tonics and a generous diet for some weeks, and sent him home to gain strength, and grow a new tibia.

He had likewise disease of the humerus, close to the elbow-joint, which I feared might be implicated. During an absence of some months exfoliations were thrown off, and the arm got well.

"Sisters." Guarding the ankle with a thick pad, I attached a bag with several pounds of sand. The cord, by which the bag was suspended, passed over a sheave placed in a stanchion, which stood in the front of the bed. Nothing can succeed better than this mode, which I have used ever since.'

As far as I could learn, even the long straight splint was then unknown in town, although Mr. Fuge, of Plymouth, told me, long after, that he had used it (with a perineal cat's tail, I believe) ten years before myself. He had taken charge of French prisoners, and learnt it from their surgeons.

If this splint approaches the axilla, as Liston directs, it hinders respiration, and will not set close to the thigh. If it rises above the ilium it restrains the motion of the body, which I preserve. My patient can sit on a shallow vessel. He can, when the weight has pulled him to the bottom of the bed, lift himself back again with his own hands. I get rid of the perineal band, which is irksome and nasty; and as the patient can shift his place, and be safely moved from one bed to another, there is little risk of bed-sores. The ankle and the heel, however, require care.

I introduced this method into the Devon and Exeter Hospital in 1841, but my colleague, Mr. James, had already invented a very ingenious apparatus with the same intent. In fractures of the upper third he combined it with Earle's bed. A model of this apparatus was exhibited in 1851; but an account of it had previously been published in vol. viii. of 'The Transactions of the Provincial Medical Association.' I have kept to my own, because any village carpenter can make it, and because I think it requires less attention, which is important in the country, where the patient and his surgeon may live miles apart. Possibly there was another motive, of which I was hardly conscious: it was my own invention, and not another's.

A callus, sufficient to replace the tibia, had also formed. The upper third of the dead bone lay exposed. The lower could be felt through sinuses.

June 11, 1862.—I trephined the new bone, nearly half-way down, enlarged the opening upwards with bone forceps, and extricated one very large sequestrum, and several smaller. Then I reflected the skin on the lower third, trephined two inches above the ankle, enlarged with bone forceps, and drew out another large sequestrum. The integuments and the new bone at and below the middle were not touched; but fragments were cleared out. The medullary artery, bleeding in jets, was stopped by a compress.

Both cavities granulated and healed, except one very small fistula near the knee. This put him to no inconvenience, and, as his health was re-established, I considered him cured on September 21, 1862.

XXXIV.—*Necrosis of tibia—Operation postponed to grow a new bone.*

Charles Sim, of Brixham, æt. 12, came to the hospital for necrosis of the tibia. As the new bone did not support the limb, I left the sequestrum, which was seven inches long, to serve as a splint, and postponed the operation for a twelvemonth. By that time the leg was firm, and the necrosed bone lay exposed; except towards the lower end, where, for three inches, it was bridged over by a sheet of new bone, covered with skin.

July 20, 1867.—The skin was cut through, and the new bone chiselled away. The sequestrum was then taken out, but it required force. A smaller fragment, penetrating into the upper epiphysis, was drawn out.

The leg required no artificial support. He was well fed, recovered his health, and went out on October 16, with the wound nearly healed.

I have had cases like these, which I need not describe; but the next shows how one may be deceived as to the sufficiency of new bone.

XXXV.—*Necrosis of tibia—New bone incomplete.*

George Davey, of Shobrook, æt 13. The tibia, between the upper and lower epiphysis, died four months before, after acute periostitis. The upper half of the sequestrum was

bare, the lower covered with skin and with new bone, as yet soft and flexible. The sequestrum was so engaged in the epiphysis, so propped by another sequestrum which lay behind it, and its lower end so bound down by skin and new bone as to render the leg quite firm. It was assumed, therefore, that the new bone was completed. But the shaft of the tibia had split lengthwise into two pieces, the outer and longer concealing the inner; and, when this latter was taken out, the new case of bone was found so imperfectly ossified, that it could not be trusted to support the leg.

November 23, 1867.—First I reflected the skin from the edges of the exposed half of the sequestrum, and then divided and reflected it from the lower half, and from the new bone. This new bone I chiselled off. Then I trephined, and cut the sequestrum in twain, wrenching out first the upper half, and then the lower. Thus the posterior sequestrum was brought to sight. Where necessary it was gouged and lifted, smaller fragments were then taken out, as far down as the lower epiphysis. Nothing was known of this inner sequestrum until that in front had been removed.

Several arteries were tied, but there was a disposition to bleed, and the wound was left open until the next day, when bread poultices were applied. As the new bone was imperfectly solidified, the leg was put in a McIntyre splint.

February 11, 1868.—The tibia was by no means firm at the upper epiphysis, so I sent him into the country for change of air.

26.—His appearance had improved—he had evidently been well fed, as he had been in the hospital; but as the tibia was no better, I ordered syr. ferri phosph. c̄ ʒj. twice a day, and sent him back.

April 6.—The bone was thoroughly united. He was ruddy and fresh, but the wound, though small, was foul, and the skin excoriated—indicating domestic neglect. I took him into the hospital again, and gave up the McIntyre splint. He was quite well by the end of the month, treading firmly. The muscles are assuming size, shape, and power. A small indolent ulcer remains.

Fistulous tracks, not depending on sequestra, will remain long after these operations. The patients are very indifferent about them, as they occasion little annoyance.

Of the three following cases, the first has peculiarities of its own; the second is not without interest; and the last, in

which the peroneal artery was tied, comprises an operation not frequently performed.

XXXVI.—*Ligation of femoral artery for secondary hæmorrhage.*

Robert Perry, of Colyton, æt. 55, came to the hospital with an oval tumour, rather less than a walnut, on the posterior tibial nerve, just above the ankle. Producing pain and numbness, it was supposed to be a neuroma; but as its removal would have paralysed the foot, I temporised, directing iodide applications. Patient and nurse both insisted that it grew less. I did not think that, but it certainly did not increase; so I sent him home, with directions to come up occasionally.

Six months after he was sent up in haste, in consequence of profuse arterial hæmorrhage. The tumour and coagulum now filled the space from the tibia to the fibula, distending the fascia, and lifting the tendo Achillis. I cut into it, and found a colloid cancer, connected with the periosteum of the tibia, and involving the posterior tibial tendon, the flexor tendons, the nerve, and the artery. It was agreed that I should amputate above the knee.

The amputation, which I devised some years since, was as follows: I took the principal flap from the front of the knee, as in resection, sweeping round the tuberosity of the tibia, and taking out the patella. Then I transfixed; made a short flap out of the skin covering the ham; divided, circularly, the soft parts at the condyles, and sawed the femur just above the epiphysis. Thus the two nerves and the popliteal vessels were cut through perpendicularly; the bearing of the femur would be on its broadest section, and the stump faced by sound integument.

My patient was a fine cheery old fellow, but he was sadly shaken. He had suffered repeatedly and severely from gouty dyspepsia. He had lived hard; sometimes working as a shoemaker, sometimes as a fisherman, which he preferred; drinking spirits whenever he could get them, and consoling himself with a young wife.

The flaps, which were attached to each other by sutures, united at once—the upper flap covering the broad end of the femur admirably; but, on June 6, a deep-seated tumefaction, not very hot, or painful, or defined, occupied Scarpa's triangle. A spirituous Goulard's lotion was applied, with diminution of size and redness. The ligature on the popliteal had not yet

separated when, June 15, a very formidable arterial hæmorrhage gushed out by its side.* As the wound was healed, except at this spot, I would not reopen it; neither, looking at the tumefaction, would I cut down on the femoral artery above the sartorius. So I tied the artery in Hunter's canal—as I had thrice before, successfully. Indeed I saw no practical objection.

His stomach had been painfully flatulent since the amputation, and so irritable that scarcely any food would stay. His urine, too, was loaded with purpurates. He was greatly exhausted by the hæmorrhage, and his stomach became, if possible, more flatulent and irritable than ever. The stump never gave way, but a copious discharge, purulent and sanious, escaped alongside the ligature. After this the swelling in Scarpa's triangle subsided. It suppurated, I suppose, and the matter came down through the sheath of the artery.

July 5.—There was cellulitis of the whole thigh.

10.—This diminished, but erysipelas extended to the shoulders. This also subsided, and he so far recovered, after remaining for several weeks in a most critical state, as to be made an out-patient; with a stump of excellent form, and perfectly healed.

I saw him March 21, 1868. He has an admirable stump, with a broad bearing on sound skin. He has not felt so well for years—which his appearance confirms.

XXXVII.—*Popliteal (diffused) aneurism—Ligation of femoral artery.*

William Thomas, of Ottery St. Mary, æt. 45, a fine active man, and young looking for his age, had a large aneurism of the popliteal artery, which was rapidly extending to the perforation of the triceps. He ascribed it to a wrench of the foot three weeks before.

December 15, 1866.—I cut down upon the femoral artery, and tied it just before it passed under the sartorius.

Nothing noteworthy occurred. The ligature came away on the thirteenth day. In five weeks the aneurism had entirely disappeared. He got up, but made too free. There was swelling, and hardness down through the canal of Hunter, as

* In this article, and elsewhere, I have spoken of tying the popliteal artery. Although quite justifiable under circumstances, yet, on the whole, I find the liability to hæmorrhage greater. But generally I tie the femoral artery as near the sartorius, whether above or below, as I am able; for all which sufficient anatomical reasons may be given.

though the sheath was full and tense; but not much pain or much heat. He was rather feverish. A Goulard poultice was laid over the artery. Suppurated slowly, and the matter finding its way upwards was discharged at the lowest point of the incision, where it had not quite healed. After the suppuration was established, the course of the swelling was smeared with ung. plumbi iod., and linseed meal poultices were applied.

March 10, 1867.—The discharge had ceased, the wound had healed, and the swelling was gone.

20.—He was again allowed to get up and move about the ward with greater care.

He was discharged quite well. He has left this part of England, and I have heard no more of him.

XXXVIII.—*Ligation of the peroneal artery.*

Daniel Baker, of Cullompton, æt. 28, a healthy labourer, was wounded, whilst mowing, by a scythe, which was unusually sharp and narrow. It entered transversely, cutting the integuments between the lowest points of the bellies of the gastrocnemius, and penetrated up through the calf of the leg to the ham. Severe arterial hæmorrhage had been controlled by a tourniquet ere he was sent off to the hospital. My first impression was that the posterior tibial artery was wounded; but the sole of the foot preserving its sensibility showed that the nerve had escaped. I found also that the soleus was not cut through.

June 27, 1866.—I passed a female lithotomy staff as a director nearly up to the head of the fibula, kept it towards the outer side of the leg, and slit open the wound. The posterior tibial artery had escaped, but the peroneal was wounded two inches below its origin. This was tied. The man had lost a great deal of blood, and was very faint, yet whenever the tourniquet was slackened profuse venous hæmorrhage followed. I could not find the orifices of these veins (for there seemed to be several in a deep hollow), but they probably belonged to the posterior tibial artery. The man's condition becoming critical, I filled the wound with graduated compresses, and secured them by fillets buckled round the leg, tighter below over the bleeding veins than above. I could thus make more pressure than with a many-tailed bandage, and, in case of need, could take off the apparatus more readily than if I had used a roller.

The patient was very exhausted, and for some time required

stimulants. When pus and sanies, with the usual fœtor, appeared, the fillets, except the lowest, were unbuckled, and the outer compresses lifted gently off. In a few days the rest separated without any bleeding. The cavity filled with granulations, the divided muscle reoccupied its place, the wound healed, and he has as good a leg as ever.

That an incapacity for menstruation should consist with female health is a strange fact. In two of the three following cases of wombless women the defect was congenital.

XXXIX.—*Removal of the womb by ligature.*

About nine years since I removed the womb by ligature. In the 'Address in Surgery' of 1860, I gave this case very circumstantially. The main facts were that the patient was 21; that it was her first labour; that she was delivered while standing; that the midwife tugged at the navel string; that the womb was everted; and that she nearly bled to death on the spot. After many months, when she was utterly exhausted by recurring hæmorrhage, I tied the womb with Gooch's canulæ.

As I was going into the operating room, the nurse, a worthy old woman and a favourite, said, 'Please, sir, if you take out her womb, I suppose she can never have her courses again, and if so, what then?' I replied, 'Nurse, if I do not take out her womb, she may die before night. If I can, I will save her life now. That is my present thought. I cannot look beyond that.'

After all manner of perils she made a thorough recovery. Of course she has never menstruated since, but when I saw her, three years after, she was in good health and good spirits. I know that she was perfectly well several years after that. Her whole aspect is feminine, her complexion is fair and delicate, as it always has been, but in no wise chlorotic. She walks with her young man like any other servant girl; she cares nothing for the loss of her womb, which seems to have produced no other effect, moral or physical, than the cessation of her menses. That is the answer to the nurse's question, 'if so, what then?'

XL.—*Deficiency of the womb—malformation.*

About four years ago a very delicate young woman, æt. 25, evidently in declining health, came under my charge at the

hospital. She told me she had been left an orphan (I believe one at least of her parents had died of consumption), that she was delicate, and had not been able to retain her water like stronger girls; but that she had been brought up carefully, was now the mistress of a national school, and had married at 23. She then added that coition had from the first been accompanied by pain, which instead of diminishing, as she expected, had increased, until she could not bear it; and that her urine escaped from her involuntarily. She and her husband, who were reputed to be very worthy young people, and a most attached couple, were greatly distressed, as they well might be. I learnt then also that she had never menstruated.

Her pudenda were perfectly natural. About three inches up the vagina there was a sudden narrowing, like a stricture. Its circular edge was soft, and the orifice sufficiently open to admit the finger; but it could have been easily dilated to a greater extent. It exerted a gentle compression.

This was the sphincter, and beyond it was the bladder. There was no trace of a womb.

I told her that sexual intercourse must cease for ever. She was much grieved on her husband's account; of whose regard for her she spoke with genuine feeling. I recommended tonics, and she went home.

I heard nothing of her until lately, when she and her husband called on the nurse. They appeared a fond couple still. Her health was restored; and the incontinence of urine not worse than when she was a child.

XLI.—*No womb—congenital defect.*

I was consulted in the case of a lady's maid, æt. 40, who, previous to the last six weeks, had enjoyed excellent health. She was a neat little figure, a brunette, must have been pretty, and was young looking for her age. She had a swollen knee, and flying rheumatic pains, for which she could not account, but she was chiefly harassed by an irritable bladder. Micturition was frequent, scarcely to be restrained, and painful both at the time and afterwards. She had used a hip-bath and opiate fomentations with partial relief. I was to ascertain if a urethral wart or a calculus occasioned her distress.

Her urine had always been apt to escape when she coughed,

but she had not been subject to leucorrhœa, and she never menstruated.

The external appearance of the genitals had nothing peculiar. The pubes and the labia were natural; the glans of the clitoris and the nymphæ were fully developed; but I could not find the urethra.

The vagina was such as might be expected in a virgin, except that there were neither transverse rugæ, nor hymen, nor carunculæ, nor fossa navicularis. It opened abruptly, just in front of the perineum. It admitted the finger, which, when it had penetrated somewhat less than three inches, came upon a dimple. This yielded, circularly, on gentle pressure, and the finger entered the bladder. As it was withdrawn the sphincter closed again. The bladder was not set on obliquely like a womb; but quite at the end of a canal, which was no vagina at all. It was simply a preternatural urethra applied, as in the male, against the rectum. There was not the slightest trace of an os uteri or cervix—neither could any womb or corresponding body be felt from the bowel.

She told me that, some years before, she had been engaged to a young man, who died. Spoke of him, as a woman should, tenderly and regretfully; but did not affect to represent her love for him as Platonic.

Judging from these cases, the menstrual discharge is not necessary to the health of a woman who has no womb. In the intervals between one pregnancy, or lactation, and another it would appear that the presence of a womb has a special and accumulating influence on the constitution; and that it is thus the organ which, by its own periodical discharge, affords that relief to the whole frame, of which it has itself created the necessity.

It is the womb alone, and not the whole generative apparatus, or, indeed, any other part of it. Looking at their personal appearance, their views, manners, and habits, and their womanly, albeit their correct and estimable feeling towards the men they loved, I believe the subjects of the cases XL. and XLI. to be real women—saving their wombs. If it be contended that their ovaries may likewise be wanting, I can only reply that, in case XXXIX. the generative system was undoubtedly complete, for that woman lost her womb, and nothing else, after bearing a child; and yet she is as healthy, without menstruation, as the two others, who never had a womb.

The following case presents such rare incidents, that I should ill convey my own impression were it embodied in a simple sketch.

XLII.—*Ruptured bladder.*

Thomas Small, of Exmouth, æt. 28, a mason, a strong and healthy man, was thrown down violently in a scuffle. His bladder was probably full, as he had been drinking. Instantly pain and a desire to make water were urgent. He tried, but could not. A catheter, next morning, only drew off a few drops of bloody urine. In the afternoon he was sent up to the hospital.

He was in great distress. The abdomen was swollen from the pubes to the epigastrium. The swelling was diffused; not rounded and defined, as in distended bladder. There was no tympany anywhere, but fluctuation everywhere; yet there was no swag, as in ascites. It was clearly a stratum of some depth, underlying the abdominal parietes. He could lie on his back, or on either side, without increase of pain; but bent his legs, on account of the distention. He did not wince on pressure. I concluded that the bladder was ruptured outside the peritonæum.

January 7, 1868.—At 4 P.M., I cut through the linea alba for two inches, from the brim of the pelvis upwards, and nicked the pyramidalis on either side. I found a large pouch behind the symphysis. The urine welled up from it copiously —for therein were extravasated the contents of a full bladder, and the subsequent secretion of twenty hours. It was simple urine, slightly tinged with blood, of which small clots came away in the catheter.

9.—The pain and excitement were diminished. The urine flowed off freely; he was directed to lie prone, at times, to favour its escape; and to take effervescing salines and diluents.

10.—Urine abundant. To omit the salines, which purged. Pulse and tongue feverish. Fluctuation scarcely perceptible. A bread poultice on the wound. He was removed to a separation ward for quietness.

11.—All fluctuation had ceased. Erythema of abdomen and flanks. Two or three ounces of urine, with a little blood, passed through the penis from time to time. To lie on water pillows. To have 8 oz. of sherry daily.

12.—The erythema had subsided. There was no tenderness of the abdomen. The pulse 90, and the tongue clean, as

indeed it continued to the last. Troublesome cough. Draining tubes were passed into the pelvis.

13.—The wound looked rather sloughy—I believe it was only soiled lymph. The urine passed through the urethra, generally; at times through the wound.

14.—Pulse and tongue right in the morning. The cough continued, with pleuritic sounds. The flow of urine decreased, but without any indication of collecting behind the peritonæum. At 9 P.M., I learnt that he had had severe rigors, and the right pleura was full of fluid. The pulse small, and 110. He was, however, perspiring, and the urine increasing. He wished to have a catheter passed several times a day. I consented—but not that it should be passed home, lest it should re-open the bladder. To take bicarbonate, acetate, and nitrate of potass in small and frequent doses, with pulvis Jacobi verus.

15.—The breathing was easier, the cough better, pulse 96. At 9 P.M., he had slight pneumonia of the left side; pulse 110. I ordered of pil. ipecac. c̄ scillâ ten grains during the night, and epithems of turpentine and mustard occasionally.

17.—Cough better, sputa less rusty, pulse 106. Urine again flowed freely from the wound. He was placed on a McIntosh sheet, with a funnel through the bed, and directed to lie on his face, when he could. At 9 P.M., he complained of excoriation on the scrotum. His bladder to be kept empty by a flexible catheter. The James's powder was discontinued, and the potass doses reduced to every eighth hour. Pulse 100. The cough was much relieved, and he felt better. His bowels hitherto had been assisted by glysters, but this day he passed solid stools.

20.—When I came this morning I found that a quantity of sloughy matter had been drawn out from behind the symphysis. I did not see these sloughs—they had been thrown away. The peritonæum was projecting through the opening in the linea alba, which had been much enlarged by the traction of the abdominal muscles; but this exposed peritonæum had not inflamed—it only showed a healthy reparative vascularity. This evening there was considerable venous hæmorrhage, apparently from the left side of the pelvis. The bleeding spot could not be seen. The salines were omitted, and gallic acid given frequently. Porter was ordered, and port wine instead of sherry.

23.—He disliked the porter, and had ale instead—a west-country prejudice. His allowance of eggs and boiled meat

was increased. A lump of plastic material appeared behind the symphysis; and the edges of the wound were varnished with lymph.

23.—His chest was well; but severe venous hæmorrhage had recurred several times.* I was shown more than a pint of blood, which gushed out in no time. I was told pressure on the pubes stopped it, but I thought it came from some vessel deep in the pelvis—it might be from the left obturator vein. As the loins were irritated by the urine a flexible catheter was kept in the bladder, and a drain-tube in the wound. As this tube was always getting out of place, and was no siphon after all, I passed a skein of thread through it, tied a bit of sponge to one end, which I put into the wound, and brought the other end into the funnel, which went through the mattress. Its capillary attraction sucked up the urine, and discharged it pretty well.

24.—No hæmorrhage, no pneumonia. I ordered a pint of wine daily and any dainty animal food which he could relish, for he was getting low; yet the bulging peritonæum still thickened and became more vascular, as if about to granulate, but all the rest of the lymph was gone.

25.—Another great gush: his strength was failing fast. I had never seen any of these bleedings, which lasted but a few seconds. Dr. Huxley was sure that pressure at the root of the penis arrested them; so were the nurses, who watched incessantly. I concluded it was so, without understanding why. A bag of ice was therefore applied, but not near the peritonæum. I could not be persuaded that such violent hæmorrhage derived its blood from the vena dorsalis penis. Traces of coagula had already led me to suspect the left obturator vein; so, that evening, I passed my finger down, and made out the obturator fasciculus for more than an inch. I felt the artery throbbing distinctly, but no aperture or clot in the vein. If I had, I could and would have tied the whole close to the thyroid foramen with Weiss's aneurismal needle. The periosteum was detached from the ossa pubis all across.

26.—An ounce of blood was lost this day. He was very weak, and as his appetite was gone the gallic acid was changed for nitro-muriatic acid and quinine.

* Dr. Huxley says in his note: 'Venous hæmorrhage, to the extent of upwards of half a pint, occurred while the patient was being lifted from the bed, and again in the evening, when coughing, to a still more alarming extent—between two and three pints having gushed from the wound in the course of a few minutes.'

27.—No bleeding. He liked his quinine and acid. Pulse 120. Tongue slightly brown, the only time.

28.—Tongue clean again. He was exhausted, and had lost heart, praying it might be over soon. He took his wine and two ounces of brandy (which, indeed, he had taken all along, except during the pleurisy and pneumonia), and his ale when he could. A bed-sore on the sacrum was imminent.

29.—A deep bed-sore. There was another feeble attempt at granulation, especially on the protruding peritonæum.

30.—I found him dying. There had been no urine secreted for some hours. He died in the middle of the day. The peritonæum remained whole to the last, still slowly thickening, and attempting to repair itself, when reparation had ceased everywhere else.

Dr. Huxley has kindly furnished a note of the examination of the body. 'Autopsy twenty-two hours after death. On a crucial section of the abdominal walls being made, no trace of peritonitis was found. Even under the exposed surface of the peritonæum there was neither lymph nor adhesion. The bladder and the portions of the peritonæum right and left of it were found completely stripped from the anterior portions of the cavity of the pelvis; the urine having formed a large sac between the peritonæum and pelvic bones, extending outwards beyond the external margins of the obturator foramina, and downwards as far as the tubera ischii. An irregular rent in the anterior wall of the contracted bladder, of the size of half a crown, communicated with this sac. The back and crests of the pubic bones were denuded of periosteum. Stretching across the sac, and forming a fold, were seen the obturator vessels on the left side, and in the vein was an ulcerated opening* two or three lines in breadth.'

I so nearly saved him that I am hardly reconciled to my failure. He was a brave man, who rendered me every assistance by his calm endurance of mortal suffering. It is easy for me, now that I review the case, to speculate on tying the obturator vein; but should I have been justified in searching, through a cleft above the pubes, for some vein or other, somewhere or other, at the bottom of the pelvis? I had indeed guessed the vein, but it was only a guess. I found it with its nerve and its artery (both of which I would have included in the same ligature without scruple), but I did not detect the

* This ulcer was probably larger than when I examined the vein six days before.

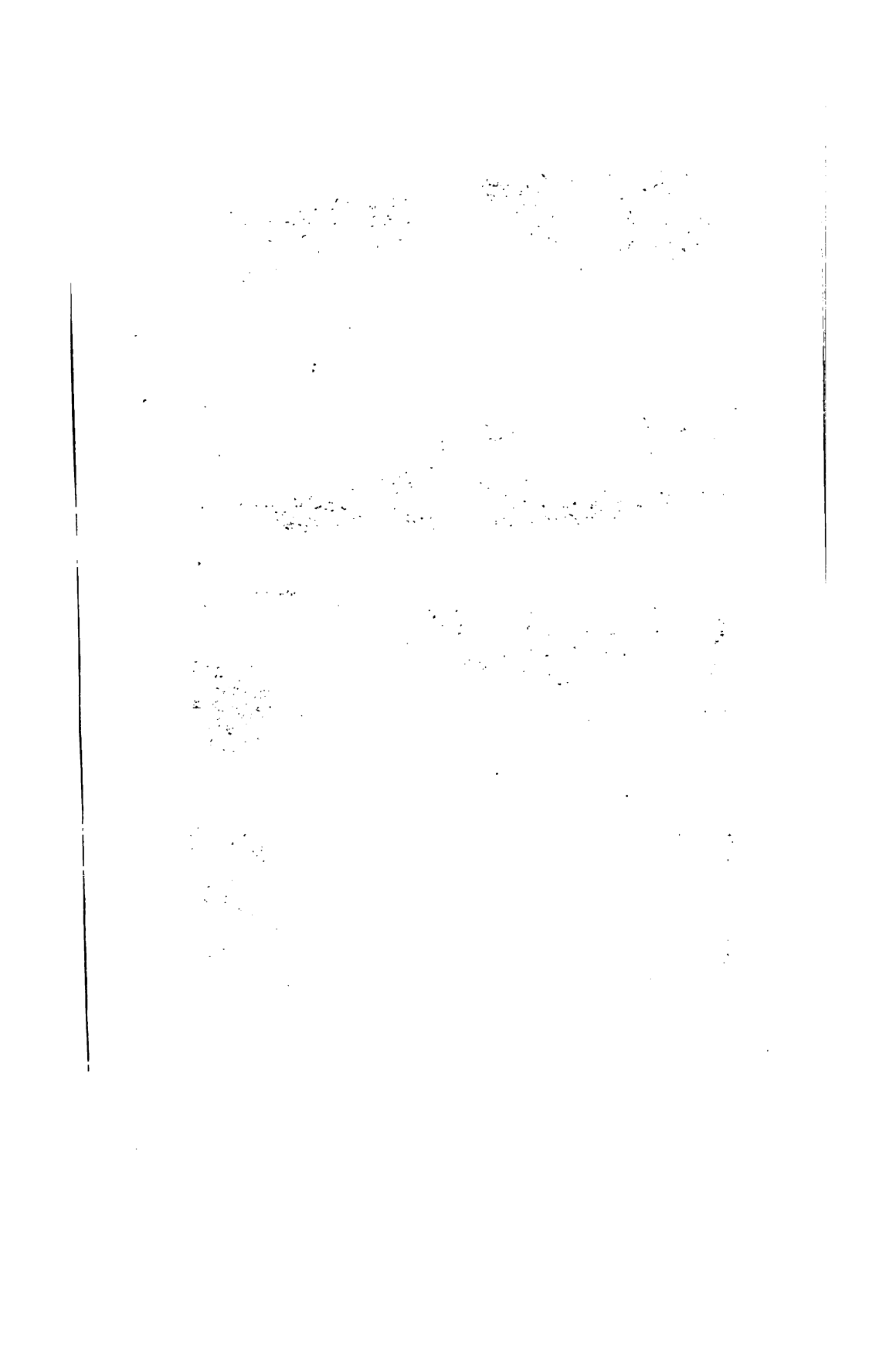


FIG. 3.

FIG. 1.

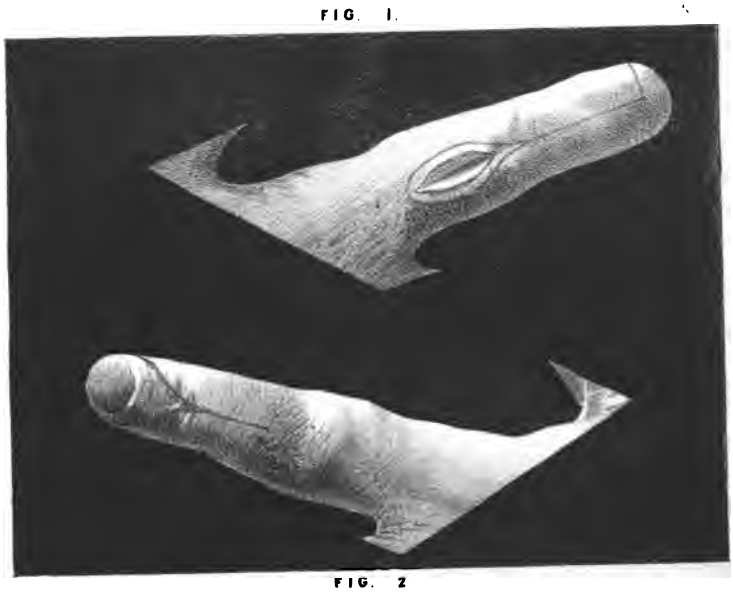


FIG. 2

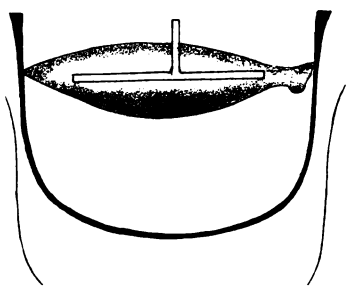


FIG. 4

FULL SIZE.

ulcerated spot I was seeking. The evidence of those who were present rather pointed to some other vein as the bleeding vessel. Above all, could I have reached the obturator vessels until the lymph were absorbed or washed away, and the periosteum and peritonæum clean gone from the pelvic bones? Yet to prolong life I would have done it even then, if assured of the source of the hæmorrhage; but that I had to learn after the event.

EXPLANATION OF PLATE I.

Diagrams illustrating amputation of the finger by swallow-tail flaps. (Case II.) Fig. 1. Front view. Fig. 2. Back view.

Fibro-cystic tumour on the patella. (Case XXII.) Fig. 3 is lithographed from a photograph of a plaster-cast. It likewise represents the tumour of Case XXIII. sufficiently to make the description intelligible.

Fig. 4 contains the plan of the incisions for removing a floating cartilage, with attachments, from the knee. (Case XXIV.) The full line represents the first incision, when the skin was drawn up. The fine line gives the position of the flap when the operation was completed. The hollow lines indicate the cuts into the joint. The cartilage is shaded. Full size.

Article VII.—*Cases of Herpes Ophthalmicus.*

By Bowater J. Vernon.

In the recent works on diseases of the skin very little attention is drawn to the importance of this variety of herpes, while in more than one no allusion to it whatever is to be met with. Indeed almost the only good account of this curious complaint is to be found in a paper published by Mr. Hutchinson in the 'Ophthalmic Hospital Reports.' *

Mr. Hutchinson's paper is based upon the observation of a considerable number of cases which had come within his own knowledge, the majority of which had been under his own care. Since its publication I have met with six or seven similar cases at St. Bartholomew's, in addition to several at the Moorfields Ophthalmic Hospital, which seem to afford some ground for comparison. It must be allowed, then, that the disease is more common than one would suppose from the little attention it has attracted. It is not a little remarkable, too, that at least four of my cases came under my observation within a few days of each

* Part iii. vol. 5

other, at a time when the weather was very severe, and that since that time, a period of several months, no similar cases have been met with. With regard to the frequency with which such cases occur it would be well to draw attention to the classification of the diseases of the cornea which Professor Stellwag* has adopted, and which, so far as I am aware, is entirely his own. A very large number of the cases which are every day to be seen at our ophthalmic hospitals, and which are termed phlyctenular ophthalmia, or pustular ophthalmia, or again strumous ophthalmia, should, according to the above distinguished authority, be considered as cases of 'herpes of the cornea,' and in very many instances the term seems a very good one as being very simple, and because the phenomena attending the occurrence and progress of these ophthalmiæ correspond very closely with those which accompany the eruption of herpes elsewhere—the severe smarting pain, the formation of vesicles on the cornea or conjunctiva, the increased flow of tears, the congestion of the ciliary vessels, and the extreme intolerance of light in many cases, all pointing to some disturbance of the fifth nerve, in all probability its ciliary branches being those especially implicated. The appearance of these ophthalmiæ in children with several forms of vesicular and pustular eruptions about the face is a matter of every-day observation.

According to this view the cases published by Mr. Hutchinson, as well as my own, would be regarded only as the more severe and more rare examples of a very common disease. In investigating my own cases I have borne in mind the points upon which stress has been laid by Mr. Hutchinson, and especially the diagnosis between the disease and erysipelas, the limitation of the eruption to certain nerve territories, and the unexplained fact that the eye itself is only involved when the eruption appears in the track of the nasal branch of the fifth nerve—at least one of my cases affords an exception to this and another seems to show that nerves of sensation are not the only nerves which suffer, inasmuch as a motor nerve, the third, was undoubtedly affected, so much so as to materially interfere with vision, and though frequent examinations were made and no structural changes in the fundus were recognised it is probable that some changes were taking place.

* 'Treatise on the Diseases of the Eye.' American translation, New York, 1868.

CASE I.—*Herpes of the Frontal, Lacrymal, and Trochlear branches of the Ophthalmic division of the fifth Nerve on the left side.*

H. G., æt. 22, a shoemaker from the Barbican, came to St. Bartholomew's February 29, 1868. He said that he had been very well until five days previously, when he felt a stinging pain in his forehead, and then some 'spots broke out' where he felt the pain, 'in the corner' of his eye, on the left side of his forehead, and lastly some few appeared on the outer half of his upper eyelid. As soon as the eruption had fully made its appearance he had no more pain except when the skin was touched. He did not feel very ill and had been able to do his work without interruption. His vision was perfect, and except some conjunctival redness the eye was not implicated.

The eruption of vesicles had apparently followed the direction of the frontal nerve, and a group of a similar character had made its appearance at the angles of the eyelids; both eyelids were swelled, as was also the cheek, but there were no vesicles on the lower eyelid, cheek, or nose. The skin of the forehead and the scalp on the right side were red and tender; the vesicles contained clear or milky fluid and were closely clustered together with bright red bases, and very painful when touched or exposed to the air.

The treatment consisted of cold lead and opium lotions to the face, and quinine administered internally. He made a rapid recovery.

CASE II.—*Herpes of the Frontal, Lacrymal, and Trochlear branches of the fifth Nerve on the left side.*

A. F., æt. 76, a feeble old woman, came to the Hospital, March 11, 1868; she had once attended at the Surgery on account of some cutaneous affection about her arms and hands, of which she was soon cured, and had felt quite well till five days ago, when she had some earache and smarting pain over the left side of the forehead and temple, and very soon afterwards an eruption appeared over the painful parts.

When I saw her the left side of her forehead was covered with the characteristic vesicles of herpes, most thickly scattered over the frontal nerve and radiating from this like the branches of a tree. The skin between the vesicles was red, œdematous, and very tender. The upper eyelid was covered with the erup-

tion; the lower lid was very puffy and swelled but was entirely free; the upper and inner part of the nose was dotted with brown crusts, the result of dried-up vesicles, but the tip of the nose was quite free. The eyesight was perfect, and the eye itself was not at all affected.

This patient did not return to the Hospital.

CASE III.—*Herpes of the Frontal and Trochlear branches of the fifth Nerve on left side, with Ulceration of the Cornea.*

A. C., æt. 22, a dressmaker, came to me at St. Bartholomew's on February 11, 1868, on account of some swelling of the left upper eyelid which had existed for two or three days. There was no very apparent cause for this, and no particular attention was attracted to it. On the 14th, three days later, when she came to the Hospital, an eruption of small transparent vesicles with bright red bases had taken place along the course of the frontal nerve, a small crescent of similar vesicles covered the inner half of the upper eyelid, and a large cluster was situated at the inner canthus. The lower eyelid was puffy, but it was free from eruption, and no vesicles were to be seen on the cheek or nose though a close examination was made for them. She had no severe pain, and did not feel ill, but her face felt 'hot and irritable.'

A cold lotion containing belladonna was ordered for the face, and sulphate of magnesia with quinine was given internally.

At her next visit, February 18, she complained of much pain in the eye: the conjunctiva was inflamed, and there was general congestion of the tunics of the eyeball, the cornea was hazy, and her vision with this eye was much interfered with.

Warm belladonna fomentations were ordered, and the saline aperient was discontinued.

In the next few days very much of the congestion of the eye was relieved, but a small depressed ulcer of the cornea was observed, with greyish borders and some new bloodvessels stretching towards it from the conjunctiva.

At this time all her pain had left her, the eruption had dried up, and no fresh vesicles had appeared anywhere. The ulcer of the cornea healed very rapidly with the employment of atropine drops and quinine in moderate doses, and in a week there was hardly any trace left of its presence.

CASE IV.—*Herpes on the left side of the Forehead, with Iritis and greatly impaired vision.**

J. S., æt. 32, a stout, healthy looking man, a discharged soldier, came to the Moorfields' Hospital June 5, 1867, and complained of an eruption on the left side of the forehead, and severe pain and loss of vision with his left eye. The conjunctiva was inflamed, and the ciliary vessels were much congested; the cornea was brilliant and natural, but the iris was discoloured, its structure was confused, and the pupil was small and irregular; oblique illumination showed considerable synechia, and a solution of atropine (gr. iv. ad ℥j.) produced very little alteration in its size. The vision of this eye was much impaired so that he could only read very large letters. (Snellen XX.)

The left side of his forehead and the left upper eyelid were pitted with red scars, like those of small-pox; those on the forehead were limited by the mesial line and did not extend to the other side. There were several of these on the left side of the tip of the nose, but both the cheek and the lower eyelid were perfectly free.

The skin between the depressions was œdematous and very tender, and he had very severe pain of a neuralgic character over the left side of the forehead and the left temple. The depressions themselves were very decidedly deficient in sensation when compared with the neighbouring parts.

He was discharged from the army on account of the loss of an arm, and had always been strong and well. Since leaving the army he had been for seven years a night watchman; when on duty a month ago he was suddenly taken very ill, and suffered very severely from pain in his forehead and over the left side of his head, and very soon after this his forehead became 'covered with a rash.' At this time his pain became more severe during several days, and at the end of a week from his first feeling ill his eyesight failed him, and his eyelids became very much swelled. He had regular medical attendance, and under treatment his pain almost left him, and the 'rash dried up,' and he came to the Hospital on account of his failing vision and occasional return of neuralgia.

Belladonna fomentations relieved his pain, but the condition of the pupil remained unaltered. The ciliary congestion was removed and his vision improved somewhat, but he remained

* This case came under my observation when I was Clinical Assistant to Mr. Wordsworth at the Royal Ophthalmic Hospital, Moorfields.

liable to severe attacks of neuralgia over the forehead, and lacrymation with ciliary pain.

After June 26, he ceased to attend.

CASE V.—*Herpes of the branches of the Ophthalmic division of the fifth Nerve on the right side, with partial Paralysis of the third Nerve on the same side.*

H. D., æt. 53, though much older looking, a philosophical instrument maker, came to St. Bartholomew's March 3, 1868, and gave the following account of himself: He had been travelling during the late severe weather through the south of England in search of work, and about a month ago, after being much exposed, he was seized with a smarting pain in his forehead and in the right side of his head; this was followed by an eruption over the painful parts and so much swelling as to close his right eye.

He came back at once to his home at Clerkenwell, and as long as his means allowed it had had medical attendance at home. He was told he had erysipelas.

The right half of his forehead and of the scalp, for the hair had been shaved off, was œdematous and disfigured by a large number of scars or pits, scattered irregularly, but much more thickly clustered over the eyebrow than elsewhere. The upper eyelid and the right side of the tip of the nose were marked in the same way, but the lower eyelid though swelled was quite free. In no instance had the scars appeared on the left side of the mesial line, and they were limited on the scalp to the part anterior to a line drawn across the head from behind one ear to the other. The scars were of a deep red colour, the skin between them being congested and œdematous, and exceedingly tender, while the scars themselves were almost wanting in sensation when touched with the point of a pen. At times he suffered intensely from neuralgic pains which never occurred to him before this present attack of illness.

The right eyelid drooped, and the eye was to a slight extent divergent, owing to very imperfect power over the internal rectus muscle; the pupil was irregular and rather dilated—it was very sluggish, but apparently free from adhesions. He complained of loss of vision with this eye; but with a very careful examination of the fundus with the ophthalmoscope I could not detect any structural changes; his vision was considerably impaired, but it was improved by the use of a

perforated card, and closing the left eye. He could read nothing smaller than Snellen XII.

He had always been an active and healthy man, with the exception of pains about his limbs occasionally in winter, which seemed to be rheumatic. As he seemed much broken in health he was admitted into Pitcairn Ward under Mr. Holden. He was an inmate of the Hospital for some weeks, and his general health was almost restored, though for some time his sufferings from neuralgia were extreme. These were at last relieved by the internal administration of quinine with the employment of aconite liniment and subcutaneous injections of morphia in the temple.

The state of the eye remained almost unchanged, except that the ptosis became much less apparent; the divergence still persisted, and the eye was practically of very little use to him, though repeated examinations with the mirror revealed no very decided cause of amblyopia.

It is probable that cases of this kind have often been mistaken and considered as cases of erysipelas, but with ordinary care there should be but little room for such an error of diagnosis, at least in the more typical and less severe instances of the kind. In the last of the cases mentioned in this paper, by far the most severe one, and in which swelling was a very prominent feature, it seems that the mistake was made; and yet the eruption, so characteristic of herpes, and so different from the bullæ of erysipelas in their size, their number, and above all in their arrangement according to some definite plan—an arrangement which generally coincides very closely with the ramifications of certain nerve branches—and their limitation to one spot would have been sufficient to point out the true nature of the case. My cases do not bear out the observation of Mr. Hutchinson that old people are more liable to be affected than the young. In every instance the eruption was limited to one side; in four of my cases the left side was the one affected, and the mesial line was never transgressed; the branches of the fifth nerve which were most severely implicated, were the frontal and trochlear, and to a less extent the lacrymal. In every case the upper eyelid was affected, but the cheek and lower eyelid invariably escaped any eruption, though generally tender and œdematous.

The first and second cases call for no special notice, as being ordinary but well-marked examples of the disease. The third of the series was to me one of the highest interest, as it afforded me an opportunity of watching its different stages.

Swelling of one or both eyelids is so common an attendant on trivial eye-affections, that in this instance there was no reason to suppose it would usher in the remarkable train of symptoms it did. The eye itself did not inflame until the eruption was beginning to decline, and the occurrence of mischief to the eye itself without any affection of the nasal branch of the fifth nerve is an exception to the rule which has been suggested by Mr. Hutchinson, that the two phenomena are closely dependent on each other. Still, on the whole, my cases support the observation, for in the two first cases the nasal nerve was not involved, and the eye escaped, and in the two last the nasal branch suffered with its fellows, and there was severe damage done to the eye itself.

It would seem that neuralgic pains of a very severe kind are a characteristic feature, and with them a very decided anæsthesia of the scars themselves, no doubt owing to the destruction of the skin. The connection between the nasal nerve and the nutrition of the eyeball must be by means of the ciliary ganglion, and this being so, it is probable that though the stress of the mischief may fall upon the cornea or upon the iris yet the disorder is much more widely felt, and it is only reasonable to expect that there would be great impairment of vision. Thus, in Case IV., though the iris seemed to be the principal seat of inflammation, there were probably much more widely-spread changes; and in the last case, which was in every way the most severe of all, it is almost certain that the amblyopia was due to nutritional changes in the nervous coat of the eye, although I could not be certain of their existence with the ophthalmoscope. This last case has one very important feature of its own, that the motor nerve of the eye was affected at the same time as the fifth. The partial paralysis of the levator palpebræ and of the internal rectus muscles was not to be mistaken: it may be that this was only a curious coincidence, and that it had no immediate connection with the herpes of the sensitive nerve supplying the same parts. Paralysis of one or two muscles of the eyeball occurring to elderly people, especially when liable to rheumatism and exposed to weather and fatigue as this patient was, is exceedingly common, but so far as I am aware has not been found to be associated with an affection of the sensitive nerve branches at the same time.

Article VIII.—*On Lithotomy and Lithotrity.*

By Holmes Coote.

THE amount of suffering caused by the presence of a stone in the bladder varies considerably: much depends on the temperament of the patient; much more on the size, weight, form, and comparative smoothness of the stone; much also on the fact of its being encysted, embedded in the ureter, or otherwise rendered immoveable; but under all circumstances its presence is a source of inconvenience, often of absolute agony to the patient; while to the surgeon's mind it presents the probability of a train of pathological events which must terminate, if unrelieved, disastrously to the patient.

No wonder, then, that so much has been written on the subject, so many schemes devised and instruments invented for the removal or destruction of this hard and irritating body. Chemical analysis has enabled us to ascertain the source whence the material is derived and in many instances to put a check on its increase; but still the fact remains the same, that in a certain number of cases we have to deal with it as an urinary concretion within the bladder, whence its removal must be effected with as little risk to the patient as possible.

The following communication does not profess to be a resumé of that which has been written on the subject, but rather to show the general experience which has been gained during the last twenty-five years at St. Bartholomew's Hospital. The enquiry also involves the interesting question how far either human suffering or life has benefited by our modern methods of operating.

In opposition to the opinion of an eminent authority who believes that calculi may form primarily within the cavity of the bladder 'by the aggregation of some sabulous matter, around and upon which fresh deposits take place until a true calculus is formed,'* I see no reason to question the more generally received opinion, that the nucleus is first formed within the lobular structure of the kidney. Its minute size, compact character, and usually simple composition is constant; while the surrounding layers, of various hues, which indicate its subsequent enlargement by concentric superposition, point to a some-

* 'Science and Art of Surgery,' by Erichsen, p. 372.

what different condition, such as would be produced by increase within the cavity of an irritated bladder. There are many specimens which show numerous small calculi within the kidney; also many cases on record in which small calculi drop during life from the kidney into the bladder from year to year, attended with symptoms which the patient himself recognises and explains on this supposition only. There are also instances of a single calculus with a double nucleus formed by the early aggregation of two distinct deposits. These, however, are not matters of immediate surgical importance.

Respecting the chemical composition of calculi, by far the greater number consist of a nucleus of lithic acid with concentric layers of lithic acid and of phosphates. But the catalogue of the Museum of St. Bartholomew's Hospital would give no true estimate of the relative frequency of such calculi, so many are thrown away or used for various purposes immediately after operation. The 'calculus of infancy' is lithate of ammonia. Next to the lithates in frequency comes the oxalate of lime calculus, rough, heavy, and compact, and usually a source of great bodily suffering, the patient's health being otherwise good. Yet a large oxalate of lime calculus may form in the bladder without pain.

A young artilleryman, belonging to one of the batteries, in good health and on his usual duty, was thrown one morning violently forward in his saddle by his horse, a powerful animal, on first leaving the stable; he felt something give way within him, and he experienced sudden and severe pain in the region of the bladder, attended with pain in micturition, a sudden stoppage to the flow of urine, and other symptoms of stone. The surgeon sounded him and detected the calculus, which he successfully extracted by the lateral operation. It was a 'mulberry' or oxalate of lime calculus, the size of a small walnut, and had evidently been impacted or adherent somewhere, for the state of the patient before and after his accident with the horse was widely different—the amount of pain caused by the movements of the calculus being almost unendurable. Perhaps it was in part within the orifice of a dilated ureter, but upon this point no satisfactory evidence could be obtained. The patient's health continued good long afterwards.

The increase in the size of a calculus seems to be due, not to a continuous deposit of material, but to its successive deposit in layers. It is remarkable how many distinct layers may be counted by the aid of a magnifying glass in the section of almost any urinary calculus.

The well-known tables published by the late Dr. Marcet* agree with general hospital experience, namely, that in a given number of cases there is an almost equal proportion of children and of adults. Out of 506 cases collected at the Norwich Hospital between the years 1772 to 1816, the list was: Children under 14 years—boys 227, girls 8, making 235; Adults—males 251, females 20, making 271; total 506. In an excellent article in Holmes's 'Surgery,' the same proportion is maintained in a list of cases collected from the London hospitals. For instance: total number of cases 177, children 86, adults 91. Sir Henry Thompson has collected a yet larger list of 1,827 cases; out of which 1,116 occurred in patients between the ages of 1 to 21 years, leaving only 717 between the ages of 21 and 82.

Between the years 1860 and 1864 inclusive 71 cases of stone were admitted; and of these 39 were under the age of 25, the remaining 32 of more advanced years. In 1860 there were 21 cases, and 12 under 25 years. In 1861 there were 13 cases, and 7 under 25 years. In 1862 there were 15 cases, and 12 under 25 years. In 1863 there were 13 cases, and 4 under 25 years. In 1864 there were 9 cases, and 4 under 25 years.

It has been stated that calculi are more common in cold than in warm climates; indeed that the disease is scarcely known within the tropics. Possibly in tropical regions the diet, mostly vegetable, exercises no unimportant influence on the disease. But on these points we have no positive information. At Smyrna calculus disease is known; among the negroes in the southern states of America it is not uncommon; in some parts of India it prevails to a great extent. Mr. Callender lately called my attention to a collection of 90 calculi removed by operation during a space of two years from patients, natives of the Punjaub, by one surgeon, namely Mr. Kiernander, formerly student at St. Bartholomew's Hospital and now in practice in India.

The mortality after the lateral operation varies according to age; or perhaps it would be more correct to say, according to the general healthiness of the internal organs, especially the kidneys. In children the mortality is about 1 in 18; in adults 1 in $5\frac{1}{4}$. At St. Bartholomew's Hospital, from 1863 onwards, there were 23 consecutive successful cases of lithotomy in patients between the ages of 2 and 20. In 8 cases of adults there were 2 deaths, or 1 in 4. One patient died from

* 'On Calculus Disorders,' p. 33.

peritonitis; the other from collapse, the kidneys being greatly diseased. This last patient never rallied from the operation. He could not take nourishment, nor respond in any way to the care bestowed upon him.

It is reported that Mr. Cheselden operated in 213 cases at St. Thomas's Hospital, of which 20 died, or 1 in 10. Mr. L. Lynn operated successfully 30 times in succession at the Westminster Hospital, and then lost 7 cases. But these statistics are of value thus far: they prove that, provided the patient be sound and free from internal, and especially renal disease, the lateral operation of lithotomy is one comparatively safe and free from risk; whereas, on the other hand, if the kidneys happen to be diseased, the chances of death are fearfully increased, and as such diseases are more prevalent in middle life, we see a ready explanation of the increased mortality at that age. I assisted the late Sir William Lawrence in private at the operation of lithotomy on a gentleman between 40 and 50. Nothing could have been more prompt and complete than the whole proceeding, and yet the patient died some few days afterwards. On examination, it was found that both kidneys were contracted and granular, and the capsules were adherent; the peritonæal cavity contained a considerable amount of thin pus.

In children the cause of death is mostly tuberculous disease of the kidneys or of the lining membrane of the bladder; or in some cases the little patients are the subjects of fits or mesenteric disease. It is stated that cases have been recorded in which death from hæmorrhage has ensued in the second week, possibly from the separation of a slough. I have not yet lost a case from such a cause, either in the infant or adult; indeed, fatal hæmorrhage seems one of the most uncommon events after this operation.

In the valuable statistical returns of St. Bartholomew's Hospital, now annually published, Mr. Willett gives a rather high death rate after lithotomy between the years 1863 and 1867: —

In 1863,	cases	7,	per-centage of deaths	14·28
„ 1864	„	9	„	„ 11·11
„ 1865	„	9	„	„ 44·44
„ 1866	„	15	„	„ 13·33
„ 1867	„	8	„	„ 12·5

Total cases 48, deaths 9, average percentage of deaths 18·75.

The lateral operation of lithotomy is not difficult of performance, nor is it in itself dangerous. And yet every surgeon,

from Cheselden downwards, has felt, I doubt not, more anxiety in his turn than he would care to confess to others. Perhaps the cause of this may be that it is nearly all done out of sight, under circumstances when an unexpected difficulty cannot be so promptly understood and met. The great difficulty has always seemed to me to arise from the fact of the opening into the bladder being imperfectly or insufficiently made, when the forceps fail to be introduced, or pass between that organ and the rectum.

I am not familiar with the 'hour-glass' contraction of the bladder. Whenever a delay occurs, this explanation is murmured from one bystander to another. There are anatomical preparations which seem to prove it; but still it must be rare indeed in practice. Mr. Wormald had a case of a boy in whom there was great difficulty in finding the stone after the bladder had been opened in the usual way. The supposed 'hour-glass' contraction was presumed to exist; but Mr. Wormald remembered that this lad had been in the habit of sitting with two fingers in the rectum, for the purpose of keeping the stone from sinking on the sensitive 'trigone,' and hence in course of time a pouch was formed, into which the stone had fallen. For the sake of obtaining relief from pressure on the 'trigone,' children have been know to sleep with the head downwards.

It is a remarkable fact that Mr. Wormald never performed any other than the lateral operation during the time that he was on active duty at the Hospital, and that he never lost a case.

The lateral operation is accomplished by making a free incision of the external parts, by passing the knife deeply and behind the bulb into the membranous portion of the urethra, by continuing the incision into the left side of the prostate, and by subsequently dilating that part and the neck of the bladder by the finger or some surgical instrument. An enema of warm water should always be given three to four hours before the operation to clear the rectum. Some amount of urine should, if possible, be retained in the bladder; the instruments are then more readily manipulated, while the escape of fluid shows that the necessary incisions have been effected. I do not endorse the opinion of Cheselden, who says: 'Here I must take notice, it is very convenient to have the bladder empty of urine before the operation; for if there is any quantity to flow out of the bladder at the passing of the gorget, the bladder does not contract, but collapses into folds, which makes it difficult to lay hold of the stone without hurting the bladder.'*

* 'A short Historical Account of cutting for the Stone, appended to the Anatomy of the Human Body.' 1768.

The patient having been rendered insensible by the administration of chloroform, and secured by proper bandages in the usual position, a curved staff is introduced, the straight part being held perpendicularly, the curved part pressed firmly upwards against the pubes. There is no fear of this leading, as the late Mr. Stanley imagined, to the incisions being made into the narrower part of the space beneath the arch of the pubes. The external incision is unconnected with the position of the staff, and when the groove in that instrument is opened, it should be by a deep incision, commencing about the middle of the external wound, the point of the knife passing behind as well as to the side of the bulb.

The first incision should commence at the raphé, about an inch and a quarter above the anus, and be carried obliquely downwards and outwards through the skin and subjacent fat to the tuberosity of the ischium. A free external incision cannot by any possibility do harm, while on the other hand it may relieve the surgeon of subsequent embarrassment. Next, the same knife should be passed steadily and deeply upwards and inwards towards the under surface of the symphysis pubis, where the urethra is sure to lie, the point of entry being about midway in the external incision between the median line and the tuberosity of the ischium. The knife will thus avoid wounding the bulb by passing behind it. It will likewise pass behind some arteries, which go to the bulb just below the triangular ligament. The membranous portion of the urethra having been opened, the surgeon may take the handle of the staff in his hand, and, with the same action that he depresses it, carry the knife through the prostate; or he may leave the handle of the staff to his assistant, while he follows the knife with the forefinger of his left hand. This latter proceeding is the better, unless the perineum be very deep. The surgeon can thus cut and dilate the prostate at the same time, and effects an entry into the bladder on easiest terms.

In this part of the operation the surgeon should dismiss from his mind the minuter points of anatomy connected with the study of the perineum. It is to him a matter of absolute indifference whether he divide the transversalis perinei muscle, the neighbouring arteries, or the anterior fibres of the levator ani. He knows that, first, he must strike one structure with certainty—namely, the membranous portion of the urethra; and, secondly, that he must make a sufficient opening into the bladder through the prostate without wounding the pelvic fascia, and in most cases this is effected as much by stretching as by cutting, and with far greater safety to the patient.

Now, the question arises, should this part of the operation be effected by a beaked or by a plain knife? To this I reply, every surgeon makes his way into the bladder in his own fashion. I prefer a plain knife rounded at its extremity, but bear ever in mind that it must on no account be allowed to leave the groove in the staff; and this in most cases is not difficult of accomplishment, all that is needed is coolness and a firm steady hand. The knowledge when the point of the finger is in the bladder is never mistaken by a good anatomist. The feeling is quite peculiar and characteristic. The passage of the forceps into the bladder, and the extraction of the stone are matters too well understood to need any special remark.

Strange as it may seem to some surgeons, I would not altogether lay aside the use of the gorget. In the event of the perineum being very deep, and the stone of more than usual size, considerable space may be gained by the employment of such an instrument as that used by the late Mr. Martineau, which was thin and light, and just so sharp as not to have a cutting edge.

Although the hæmorrhage, whether arterial or venous, after the lateral operation is sometimes profuse, it usually ceases shortly after the patient is untied and put into bed. I cannot remember a case of death resulting from the direct loss of blood, although in a few cases the patient has become completely blanched. One of the most severe cases commenced about the twelfth day after the operation. In the 'Edinburgh Medical and Surgical Journal' for 1831, some cases of death from hæmorrhage are recorded, and allusion is made to a so-called irregularity of the pudic artery, described by Tiedemann. The arteria dorsalis penis instead of being the termination of the internal pudic is furnished by a branch of the internal iliac, and runs close by the side of the prostate.

When the operation is completed and the stone removed, the patient should be put on his left side in bed, with a folded cloth under the nates. No tube nor other instrument should be introduced through the wound into the bladder. The patient is only inconvenienced by such a measure, and when the incisions have been properly made there is no fear from the escape of urine. It has been suggested by Mr. Wise that two-thirds of the fatal cases proceed from the fact that after the operation the urine flows over an open wound. No evil consequences have been noticed by me in leaving the urine to escape over the cut surfaces, the other conditions of the case being favourable.

The median operation, a variety of the Celsian method, or cutting on the grip, now coming before the profession, has during the last twenty-five years been frequently performed at St. Bartholomew's Hospital, and in more ways than one. I cannot stigmatise it as an operation suited only for those who have an imperfect knowledge of anatomy. But I maintain that the supposed advantages said to proceed from the division of structures of the mesial line are not yet established; that it is not specially fitted for the removal of large stones, and that the mortality, as far as we know, is as great as in the lateral operation—perhaps greater. Within this year the rectum has been wounded once, and even twice, in the same subject. I remember seeing a boy die from collapse after this operation, without apparent cause. Mr. Stanley performed this operation in the following way—the patient having been secured in the usual position, and the staff introduced, a semi-lunar incision was made, the concavity directed downwards above the anus. The finger introduced into the wound soon felt the groove in the staff, when the membranous portion of the urethra was opened and the prostate was cut either on one side or on both, or only dilated, according to the size of the stone. The introduction of the forceps and the extraction of the stone was accomplished without difficulty. Mr. Lloyd followed a very different proceeding. The patient being secured, and the rectum emptied, he introduced a speculum open above into the bowel. He then introduced a grooved staff into the bladder, and made a vertical incision from the staff into the rectum, thus throwing the urethra and the bowel into one. Next he passed through the opening of the urethra into the bladder an instrument not unlike curling tongs, by which he dilated the prostate by the separation of the blades, so as to enable him with ease to extract the stone with or without any use of the knife.

But when asked what are the advantages gained by operations different from those obtained by the lateral method, we are at a loss for an answer. Let us remember that the median operations have not the merit of novelty, and in reverting to them we are travelling in a circle and over well-trodden ground. Knowing the chief sources of danger which influence the progress of a case of the lateral operation, and viewing that operation as one in itself eminently successful (as proved by the statistics of children), it appears to me that, instead of varying the line of incision or occupying ourselves in some other mechanical detail, we should turn our attention to the

improvement of the patient's general condition, especially that of adults, or else substitute an entirely different proceeding, such as that of crushing the stone by the lithotrite, or grinding it into a powder.

I have not in these remarks adverted to the high operation. In many cases it is impossible of performance, and in none offers advantages such as I should have ever liked to avail myself of.

The operation of lithotrity, or lithontripsy is one which immediately attaches itself to the mind of both surgeon and patient. What so natural a method as to crush the stone into small fragments within the bladder. How desirable, especially in adults, to avoid the perils of a deep incision with its concomitant dangers. I grant this, and look forward to the day when instruments will be invented, one of which I witnessed many years ago at St. Bartholomew's Hospital, by which the stone will be ground to a fine powder, and escape in this form, mixed with the urine, through the urethra. The present operation of lithotrity breaks up the stone into sharp and angular fragments, and is so far imperfect. My first experience consisted in viewing the operations performed by the Baron Heurteloup, who exhibited his method of operating at St. Bartholomew's Hospital. The bed which he used, and by means of which he could depress the head of the patient, is still preserved, and occasionally in use. The following were the stages of the operation:—

1. The injection of the bladder by warm water.
2. The exploration of its cavity.
3. The seizure of the stone.
4. Its destruction.
5. The removal of the fragments.

Now, the injection and distension of the bladder by warm water, after the abstraction of the urine, was found to be a very painful and distressing proceeding and wholly unnecessary. The walls of the bladder are often thick and unyielding, and the forcible expansion of the organ causes the patient to exhibit many symptoms of distress. The Baron in vain assured the bystanders that the patient was not suffering pain; the very aspect of the man gave a contradiction to this assertion. The sweat often poured from his brow, and the face became livid and blanched. Next it was found (for I speak of thirty years ago) that with the improvement in the manufacture of the lithotrites, and with the diminished length of the curve, the instrument could be easily manipulated within the bladder without any

addition of warm water. The fear that the walls of the bladder might be injuriously included in the blades was not real. Such an event is felt at once by the patient, who could, if necessary, tell the surgeon what he had done, and moreover the surgeon would feel that he had included something within the blades of the forceps which hampered their free action and presented not the usual hard unresisting surface. I am confident that in some cases I have included a fold of bladder within the blades of the lithotrite, and should have done damage had I not been guided by both my own experience and the explanation of the patient's feelings. A very slight movement of the handle of the lithotrite serves to free the blades from such an accident. The abstraction of the urine from the cavity of the bladder and the subsequent distension of the organ by warm water has been given up at St. Bartholomew's, as far as I remember, for years, the patient giving the assurance that he had retained his water for some hours before the operation.

The exploration of the cavity of the bladder, the seizure of the stone and its destruction should scarcely be regarded as three distinct stages of the operation. They should rather be regarded in the light of one compound act, to be completed with the greatest possible amount of dexterity, quickness, and gentleness. The operation may then be repeated at rapidly following intervals without causing any great amount of distress to the patient; while, on the other hand, a prolonged operation, accompanied by rough usage, may excite symptoms which may put the patient 'hors de combat' for many weeks or months, or even endanger life.

We hear of some surgeons who perform lithotrity with more than the usual amount of success; such is doubtless due to greater delicacy of manipulation and attention to many little points on which the patient's subsequent comfort will depend. But if the question be put whether the operation is free from risk and suitable to all cases of adults, we must give a qualified reply. In this, as in all other important operations, there are attendant perils, and as regards lithotrity, they seem to increase in proportion to the size and hardness of the stone, and the general condition of the patient's health. Thus far it is the same as with lithotomy. But, on the other hand, there are cases where lithotrity may be performed with success, and where lithotomy would be almost assuredly fatal.

We cannot deny that many of the dangers following the operation of lithotrity yet remain. Others, however, are diminished or removed owing to the superior construction of instru-

ments. Thus we do not anticipate perforation nor ulceration of the mucous membrane of the bladder nor of the urinary passages; nor the fracture of the instrument, with ordinary care. But pain and general nervous disturbance may supervene, inflammation of the mucous membranes, or of the peritonæum, may arise. There may be swelling of the testicle, inflammation of the pelvic veins, retention or incontinence of urine, urinary fistulæ, or a minute fragment of the calculus may remain to form the nucleus of another stone. In 1850 a distinguished surgeon operated with the lithotrite on a patient aged 50. In about a month the patient died. The bladder was found full of pus and fragments of stone on which it was contracted. The mucous membrane was inflamed, pus flowed from both ureters, and the kidneys were extensively diseased.

Among the unpleasant consequences which have more recently come under my notice is that of chronic cystitis, attended with thickening of the walls of the bladder and the most acute pain in voiding the urine. The agony upon each recurrence of this act is such as to cause the sweat to stand on the patient's face and make him roll on the nearest couch. One patient always needed on such occasions the hypodermic injection of morphine, an apparatus for the use of which he always carried about with him. Another patient, thus affected, declared that since the operation his sufferings had been aggravated rather than relieved.

Cases have been recorded of acute cystitis preceded by rigors, terminating in death, where the bladder has been found surrounded by pus. Here, perhaps, too much violence had been used. A case was mentioned to me by my colleague, Mr. Savory, in which the surgeon, in his endeavours to crush a hard and large calculus, so firmly fixed the blades of the lithotrite in the stone that he could not free them, neither could he use greater force for fear of fracture. In this dilemma he managed by introducing two fingers into the rectum to press the stone from the grasp of the blades, and to remove it subsequently by the lateral operation. The blades of the lithotrite were found on examination twisted and injured.

Although I am not prepared to say that the hammer may be of use in breaking some hard and brittle calculi, yet it cannot be denied that the use of the screw is a great improvement. Of late some modifications of the screw have been introduced, rendering more easy, equable, and gentle the separation and closure of the blades. The great point for the surgeon to bear in mind is to keep the blades well in the centre of the

organ, and to avoid, if possible, any rough touch or injury to its walls.

Let us next ask, To which class of cases is the operation of lithotrity applicable? Although it has been performed, and with success, on young children, I think, under all the circumstances, they should be excluded. In adults we should perform lithotrity in all such cases, as where a piece of gutta percha bougie or other similar substance has slipped into the bladder and became incrusted with phosphates. In the same list should be included all those cases in which, in men of middle or advanced life and of gouty habit, a small lithic acid calculus drops from time to time from the kidney along the ureter into the bladder. I have operated on several of such cases, and one sitting has been sufficient to enable the fragments to pass by the urethra. But yet larger calculi may be safely removed by this process. Uric acid calculi of an inch to an inch and a quarter in diameter or even larger, and somewhat smaller specimens of the oxalate of lime calculi in which the phosphates predominate, are also fit for the lithotrite, being soft and readily friable.

The urethra must be moderately healthy, and the bladder free from active disease; the cases, in short, should be selected.

That the dangers from lithotrity are not wholly imaginary may be inferred from the statistics of the Hospital. It appears that the mortality from this operation is as high as 33·33 per cent., while from lithotomy it is only 18·75. But here we must correct an error, for if the young under 20 years are withdrawn the mortality after lithotomy is often above 50 per cent.

That lithotrity offers many advantages to the adult is obvious. The main object of modern ingenuity should be the discovery of some mechanical contrivance by which the stone could be pulverised, and the irritation caused by the sharp fragments entirely avoided.

The mortality after the lateral and indeed other forms of lithotomy seems ascertained with much certainty.

The median operation is by no means free from those risks which beset the lateral operation. In 139 cases of Allarton's operation of all ages, there were 13 deaths, or 1 in 11; and when this proceeding was restricted to adults the death-rate was 1 in 7; over 50 it was 1 in 2·5.

ARTICLE IX.—*Contributions to Cerebral Pathology.*

By WILLIAM S. CHURCH, M.B. Oxon.

THE following cases of disease of the brain, its vessels, and meninges, have been examined in the post-mortem room of the Hospital during the fourteen months ending July, 1868.

The difficulty of diagnosis which attends almost all cases of cerebral disease is allowed by every one to be very great. Symptoms, as, I think, these cases will demonstrate, beyond pointing to cerebral disease, are of very little use; notwithstanding the clear and definite rules laid down by many authors for distinguishing inflammation of the meninges and of the brain, tubercular from simple meningitis, apoplectic effusion from embolism and softening, clinically the difficulty remains, and an appeal to probabilities is often all that remains for the physician. Hence every case of brain disorder is worth recording in hopes that 'per viam exclusionis,' if not by the presence of distinctive symptoms, the diagnosis of some at least may be rendered more satisfactory and definite.

The comparative rarity of some of the cases induces me to think that they may be of use to those who have not the advantage of attending a large public hospital, and who consequently can but rarely meet with cases similar to some of those here recorded.

The total number of deaths from cerebral affections, during the fourteen months, is not represented by the post-mortem records, not a few bodies being removed without examination, and one instance of chronic hydrocephalus is not included among those published.

Ten cases of apoplexy were examined with the results appended in the table on page 142.

The table speaks for itself; nothing could better illustrate the connection between renal disease, disease of the arteries, and hypertrophy of the left heart. In six out of the eight cases not associated with valvular disease, the kidneys are noted as diseased to a greater or less extent. Case IV. is interesting; intra-cranial aneurism rarely occurring at so early an age. I have only been able to meet with two cases in which intra-cranial aneurism has been noticed earlier in life, Dr. Crisp* having seen a case in a boy of 14, who became giddy and insensible whilst carrying a heavy weight. June 20,

* 'Diseases of the Arteries,' vol. i. p. 165.

No.	Name	Age	Site of Hæmorrhage	Condition of Heart and Arteries	Condition of the Kidneys	Remarks
1	John W. .	31	Right optic thalamus, breaking into the ventricle	Loaded with fat externally. Hypertrophy and dilatation of left ventricle. No atheroma of cerebral arteries. W. ℥xv.	Granular. Studded with cysts. W. of the two ℥xj℥.	Liver extremely fatty. His friends stated that 'he lived on spirits.' Died five hours after first symptoms
2	*Joseph L. .	47	Frontal lobe of left hemisphere	Flabby and pale. W. ℥x. . .	Natural	No mention of the condition of the cerebral arteries
3	William B. .	58	Left side of pons. Fourth ventricle filled with blood	Loaded with fat externally. Left ventricle hypertrophied. Atheroma in aortic and mitral valves. W. ℥xv.	Very granular. Equal atrophy of the cortical and pyramidal structures. W. of the two ℥vj.	When brought to the Hospital his friends thought he might have taken opium. Died within twelve hours from seizure
4	Charles Edwin W.	15	Cavity of arachnoid. Laceration of brain tissue at apex of right Sylvian fissure	Aortic valves shortened, thickened, and incompetent. Stenosis of mitral with vegetations. W. ℥xiij. due wholly to hypertrophy of left side. Arteries generally healthy	Firm and tough. W. of the two ℥ix.ss.	An aneurism the size of a pea found on the right middle cerebral artery at the apex of the Sylvian fissure. Picked up senseless in the street. Died within two or three hours. Vomited and had a succession of fits whilst in the Hospital. Rheumatism when six years old
5	John C. .	47	Central portion of left hemisphere	Slight hypertrophy and dilatation. W. ℥xiv.ss. Atheroma of cerebral arteries	Granular. Studded with cysts. W. of the two ℥vj℥.	Admitted insensible. Died in a few hours
6	George P. .	52	Pons and crura cerebri .	Left ventricle slightly dilated. Hypertrophied. W. ℥xvj.ss. Cerebral vessels very atheromatous	Loaded externally with fat. Much atrophied, but not very granular. W. of the two ℥viij.ss.	Died in thirty-five minutes from the time that he fell insensible
7	Elizabeth P.	47	Substance of left hemisphere about the level of the corpus callosum	Firmly contracted. Valves natural. W. ℥xij.ss. Considerable atheroma of the arteries of the brain	Capsules closely adherent. Tissue coarse. W. of the two ℥vij℥.	Fell insensible as she was getting out of an omnibus. Died within two hours
8	Emma W. .	20	Lying along the outer side of ventricle. Walls broken down and ventricle filled with blood-clot	Aortic valves broken down. Calcareous, covered with vegetation. Aneurism of mitral valve. W. ℥xvj. Cerebral vessels specked with atheroma	Embolic wedges in them. W. of the two ℥viij.ss.	Fit followed by hemiplegia November 6; again on the 16th. Able to walk on the 24th with assistance; 25th, convulsions and death
9	Elizabeth A.	52	Substance of the left hemisphere. Blood undergoing absorption, decolorised	Cerebral vessels free from atheroma. Heart natural	Capsules adherent. The kidneys were far advanced in decomposition	Lived one month after the apoplectic fit
10	*Mary D. .	33	Corpus striatum and substance of left hemisphere and ventricle	Heart natural. The larger arteries healthy, but the small cerebral branches atheromatous	No abnormal condition noticed	Died within a few hours
11	—— (female)	35?	In the cavity of the arachnoid	Healthy	Healthy	Picked up by the police insensible, supposed to be drunk. Fracture of the orbital plate of left frontal bone. Hæmorrhage into the cellular tissue behind the eye, and great hæmorrhage into the arachnoid

* The post-mortem investigations of the cases thus marked were not made by myself.

1839, he recovered his sensibility, and again became insensible on July 7, and died July 11, twenty-one days after the first symptom. In this case two aneurisms were found on the right anterior cerebral artery; the larger of the two had burst. The second is recorded by Mr. Kingston * as occurring at the same age. Mr. Holmes † records a case of aneurism of the internal carotid in a boy of 16. Dr. Gull ‡ and Mr. Brinton also notice several cases occurring in early life, but intra-cranial aneurism is rare before 20. Ordinary apoplectic effusion is probably rarer still, Dr. Gull remarking, 'whenever young persons die with symptoms of ingravescent apoplexy, and after death large effusion of blood is found, especially if the effusion be over the surface of the brain in the meshes of the pia mater, the presence of an aneurism is probable.' This case also serves to confirm the truth of his remark, that aneurism in young subjects commonly occurs without disease of the vessels.

In Case VIII., in which heart-disease was present, the brain substance was so torn and broken down by the effused blood that it was impossible to ascertain the exact seat of the hæmorrhage, or the condition of the brain substance anterior to it. The state of the heart, and the hemiplegic attacks on the 6th and 16th of the same month, render it, I think, highly probable that embolism and consequent softening of the brain had occurred; then the vessels deprived of the support of the surrounding brain substance were unable to sustain the pressure of the blood transmitted to them by a vastly hypertrophied heart, and gave way, all the effused blood appearing to be of the same date. I have included in the table Case XI., because during life the patient presented every appearance of ordinary apoplexy. The head was carefully examined, and no trace of external injury was found. After death there was slight discoloration of the integument beneath the left eye; but it was not until after the skull was opened, and the dura mater, which was not itself torn, had been stripped off the bones, that the fracture was apparent. The hæmorrhage had taken place within the arachnoid cavity, and in the orbit, external to the pericranium. When first picked up by the police, she was supposed to be drunk, and was taken to the station-house. At the inquest no evidence was brought forward which threw any light on the circumstances under which

* 'Edin. Med. Surg. Journal,' vol. lviii. p. 69.
† 'Path. Trans.' vol. xii. p. 61.
‡ 'Guy's Hospital Reports,' 3rd series, vol. v.

the injury happened. Case VI. is noteworthy for the rapidity with which death ensued.

In the three next cases cerebral mischief was consequent on disease of the ear: death in two resulting from the cerebral affection, in the third from lung complications.

CASE XII.—*Disease of the ear—Meningitis—(?) Rheumatic disease of the heart—Pain in the head—Vomiting—Delirium at night—Death.*

Elizabeth L., æt. 17, admitted July 3, died July 29, 1868.

History.—Her illness commenced about nine weeks before admission with cessation of the catamenia and pains in the feet and legs, and afterwards in the wrists and shoulders. About five weeks before admission pain in the head commenced, and was followed in a few days by violent sickness. For a fortnight before admission she was usually delirious at night, and occasionally in the day. On admission she was remarkably anæmic, and somewhat wasted. She vomited two or three times daily, generally after food; very apathetic, but sufficiently conscious in the daytime to answer questions coherently though slowly. She wandered at night. Her bowels acted regularly; urine natural. Pulse always regular and rapid, though dicrotous. After she had been about a week in the Hospital the discharge from the left ear commenced. The tache cérébrale, which was well marked on her admission, was absent towards the close of life. The abdomen was generally retracted, and phantom tumour was present during part of her illness.

Autopsy twenty-eight hours after death.—Body emaciated. Rigor mortis slight. Offensive discharge from the left ear, and discharge from the vagina. Scalp and calvaria natural, the latter thin. On opening the sac of the arachnoid a little discoloured serum escaped, and both layers had on them shreds of lymph, here and there blood-stained; more of these shreds were situated on the left than on the right side. Considerable ecchymosis over the right parietal, and less on the left temporal lobe. The convolutions were not at all flattened: on the surface of the left frontal lobe, situated beneath the membranes, was a small yellowish white speck, about the size of a small pea. This, when cut through, was soft, and looked to the naked eye like pus; a similar little speck was on the right posterior lobe, and two smaller ones on

the right frontal lobe. At the base of the brain a considerable quantity of slightly turbid serum stained with blood was found. About the optic commissure the membranes were slightly thickened and opaque, no undue adherence of the sides of the Sylvian fissures, but the pia mater in them was extensively ecchymosed. On section the brain was congested, and the grey matter had a pinkish tinge. The fluid in the ventricles was similar to that at the base of the brain. The fornix and septum lucidum were firm. The velum interpositum thickened. Over the upper and anterior surface of the pars petrosa the dura mater was non-adherent, and the surface of the bone was discoloured, but smooth and not necrosed. Pleuræ were natural. No tubercle in the lungs; the bronchial glands contained cretaceous masses. There was an extreme amount of fibrinous vegetation on the mitral valve; the aortic were also studded with warty vegetations. The spleen had a broad band of firm consistency and yellow colour completely dividing it. This band was wedge-shaped, with the apex to the hilum. Several embolic wedges were found in the kidneys. The liver was œdematous. Uterus virgin. The mucous membrane of the os granular. The mesenteric glands were remarkably small.

CASE XIII.—*Necrosis of the temporal bone—Abscess in the cerebellum—Otorrhœa—Cessation of the discharge—Pain in the head—Vomiting—Impairment of vision—Death on the eighteenth or nineteenth day from commencement of acute symptoms.*

Richard C., æt. 16; admitted July 30; died August 4, 1867.

History.—A poorly nourished boy, who worked as a butcher. Ever since he had scarlet fever, twelve years previously, he had suffered from otorrhœa, sometimes from one ear, sometimes from both; the left has been more frequently and more continuously affected than the right. A fortnight before admission he thought he caught cold; the discharge from the left ear stopped, and he was soon after seized with intense pain in the ear and back part of the head. A week before admission he first vomited, and has continued to vomit occasionally ever since. On admission the pupils were dilated and sluggish, the eyeballs in constant oscillation, slight ptosis of the left eyelid. He carried his head thrown back and stiffly. Tongue thickly furred, bowels confined. Pulse 68, and rather irregular; respiration normal. Three days after admission, August 2, he was in a state of great restlessness, with tonic spasm of the muscles of the upper

extremities and of the back. He was perfectly conscious, and complained of pain in his head. On the 3rd he was said to have had a rigor in the morning, in the afternoon he was perfectly unconscious, screaming out at times. Pulse 108, feeble and irregular. Temperature 100° F. On the 4th he recovered his senses, and recognised his mother's voice, but his vision was affected, and he was unable to distinguish objects. He answered questions slowly and rationally, complained constantly of his head. His abdomen was retracted, and he passed a motion unconsciously. Pulse 108; temperature 103·4° F.

Autopsy twenty-four hours after death.—Body poorly nourished. Rigor mortis slight. Scalp and calvaria natural. The dura mater intensely congested. The longitudinal sinus contained a large quantity of fluid blood, and a small recent clot. The vessels of the pia mater were gorged with blood, and the convolutions over the left hemisphere were slightly flattened. On the upper surface of the posterior portion of the frontal lobe an opaque white spot, the size of a shilling or rather larger, was seen which felt firm and hard.

On slicing the brain the puncta cruenta were unusually numerous, the grey matter had a pink tinge, and the under surface of the fornix was softened; numerous small ecchymoses were found in connection with the vessels of the velum interpositum.

Nearly the whole of the left lobe of the cerebellum was occupied by an abscess containing healthy-looking pus, and lined by a delicate pyogenetic membrane. Between the dura mater and the upper portion of the petrous bone some very offensive pus was found, and the surface of the bone was roughened, eroded, and discoloured.

The lungs were in a state of lobular pneumonia, but there was no evidence of any pyæmic abscesses. The remaining viscera were natural.

CASE XIV.—*Necrosis of the temporal bone—Gangrenous abscesses in the Lungs—Death on the twenty-eighth day from commencement of severe symptoms.*

Amelia W., æt. 22; admitted February 5; died February 18, 1868.

History.— She had suffered from a discharge from the left ear for twenty years; this had suddenly stopped a week before her admission. On the same day she was seized with severe pain

in the head, and frequent rigors followed by sickness. These symptoms remained unabated until admission. When admitted she was very much flushed about the face, and her expression was indicative of great pain: occasionally the pain was so severe that she shrieked aloud. On the 11th her breathing became very rapid and shallow, and she complained of intense pain in the chest. Percussion was impaired on the left side anteriorly, but no crepitation or other morbid sounds were heard. On the 16th she expectorated much mucus mixed with some blood, and her breath became offensive. On the 17th her breath was most horribly offensive, and she died exhausted on the 18th. During her life in the Hospital she had no vomiting. Subcutaneous injections of morphia alleviated the pain very greatly, and she was able, until the lung mischief came on, to take a fair amount of nourishment, and pass pretty easy nights.

Autopsy twenty-seven hours after death.—Body well nourished. Discharge of fœtid pus from the left ear; rigor mortis moderate. The scalp and calvaria natural. The brain appeared quite natural, no softening or undue congestion even in the part most closely in connection with the diseased bone. The dura mater over a space corresponding to the upper and inner surface of the petrous bone, and extending backwards over the mastoid and a little beyond the occipito-mastoid suture, was separated from the bone by a quantity of decomposing fibrinous material, which looked like a blood clot decomposing and breaking down, being indistinguishable from a mass of decomposing blood-clot occupying the inferior petrosal sinus, and spreading from it down to the jugular vein, where it became continuous with an ordinary blood-clot. The dura mater was discoloured but not ulcerated; the surface of the bone looked eroded but was not much roughened. On sawing through the petrous bone, it was found slightly discoloured. The mastoid cells were the chief seat of the disease, being filled with dark, very offensive fluid, but no great amount of loss of bony substance had taken place.

On opening the thorax, the pericardium was found to contain ℥j. of slightly turbid fluid. The heart contained a few firm fibrinous clots. Both pleuræ contained a little horribly offensive fluid, which resembled putrilage rather than pus, and were covered here and there by thin discoloured lymph, which was thickest on the diaphragmatic portions of the pleuræ, and more on the right than the left pleura. Throughout the left lung were numerous small abscesses, many of which had become

quite gangrenous; the intervening portions of lung tissue were acutely congested and softened. The right lung was in a similar but more advanced condition; the middle lobe was in a state of complete gangrene. Into the broken down lung tissue of the middle lobe hæmorrhage had taken place, a considerable-sized, recent, bright blood-clot occupying the greater part of the lobe. The lower lobe was collapsed and contained a few gangrenous abscesses. The liver was œdematous and congested, but contained no purulent deposit; its weight 4 lbs. 7½ oz. The spleen soft and pulpy; its weight ℥vij. Kidneys were natural. Uterus and ovaries greatly congested.

Dr. Abercrombie narrates a case (IV.*) extremely similar to the last here recorded, in which the lateral sinus was partially filled by clot, and in this case death was occasioned by an effusion of puriform fluid into the pleuræ. During the whole illness of Case XIV., the mind was quite clear and unaffected, and after her admission into the Hospital she had no vomiting, two symptoms which contrast with the more or less constant vomiting and mental disturbance met with in Cases XII. and XIII., in which the brain or its immediate covering was involved. I was unavoidably prevented from examining the small pus-like specks present in Case XII. microscopically; to the naked eye they had all the appearance of pus, and the absence of any evidence of embolism in the brain itself renders it improbable that they were the result of embolism of the vessels of the pia mater. These three cases together form an instructive series of the different modes in which disease of the ear may terminate fatally: 1st and commonest, by abscess within the brain itself; 2ndly, by pyæmia and pyæmic and gangrenous abscesses in the lungs, which especially occur in connection with thrombus of the cranial veins or sinuses; and, 3rdly and more rarely, by acute inflammation of the arachnoid and pia mater, leading to the effusion of lymph, serum, and occasionally pus.

CASE XV.— *Abscess in the left cerebral hemisphere—Impaired health for nine months—A 'fall' into the water five weeks before death—Lassitude, fretfulness, vomiting, coma, and death with very slight pain in the head.*

Henry C., æt 15; admitted September 20; died September 27, 1867.

History.—A scrofulous-looking lad, who worked as a butcher.

* 'Pathological Researches on Diseases and Disorders of the Brain and Spinal Cord.' Third edition, p. 35.

His health, according to his friends' account, had been failing since Christmas 1866. Five weeks before admission he fell into some water and was nearly drowned,* and very much frightened. Soon after the accident he complained of more than usual lassitude, became very fretful, and frequently vomited after meals.

On admission he appeared in a nervous, agitated state, and complained of slight pain in the lumbar region; though feeling very weak he was able to walk without assistance upstairs to the ward. His tongue was clean, but he had no appetite, and the bowels had not acted for seven days. His urine contained a large amount of phosphates and indican, but no albumen or sugar. Pulse 64. Ordered an injection of warm water.

Sept. 21.—The bowels had acted freely, some hard fæces and scybala coming away. On the 22nd partial loss of power and sensation on the right side was noticed; most marked in the upper extremities, and in the fifth pair of cerebral nerves. On the 23rd the pulse was 56 and irregular. On the 25th he first made complaint of pain in the head. Pulse irregular, and varying from 56 to 68 per minute. On the night of the 26th he was restless. On the 27th the pupils were dilated and divergent strabismus was noticed. During the 27th he became comatose and died without again becoming sensible.

Autopsy thirty hours after death.—Body fairly nourished. Rigor mortis moderate. The genital organs were undeveloped. The testes, which together weighed only 88 grains, were situated at the external abdominal rings. The scalp, especially near where it had been blistered, was œdematous; it separated readily from the bones of the skull which were very thin and almost transparent. The dura mater was greatly congested, and the lateral sinuses filled with fluid blood. The convolutions on both sides of the brain were flattened, but the flattening was most marked over the left hemisphere. On slicing the brain, an abscess about the size of a pullet's egg was found in the centre of the left hemisphere. The abscess was limited by a dense, tough wall, lined on the inner surface by a delicate pyogenetic membrane, and contained pus of deep green colour, which had no offensive odour. The brain substance round the abscess was very slightly, if at all, softened;

* Dr. Ogle, in the 'British and Foreign Medico-Chirurgical Review, No. 70, case 92, records a case in which a somewhat similar history is given—a woman was exposed to the sun, and soon after fell into some water and was much frightened; two weeks afterwards she was admitted into the hospital with cerebral symptoms. Her health had not been good for some months previous to the accident.

and the wall of the lateral ventricle in close contiguity with it appeared quite natural. The corpus striatum and optic lobe on the left side were softer than the corresponding structures on the right.

The dura mater appeared everywhere healthy, and no disease of the bones of the skull or ear could be detected.

The wall of the abscess, when examined microscopically, was found to be made up of dense fibrous tissue, and presented no appearance of lamination, no hooklets of echinococci or cœnuri were found.

The remaining viscera appeared healthy. The kidneys were in a state of intense congestion, and the blood throughout the body was fluid.

Drs. Gull and Sutton, in their recent article on abscess of the brain, in Reynolds's 'System of Medicine,'* are reluctant to admit that cerebral abscess ever occurs idiopathically; remarking, that often the 'primary disease was so small that it might easily have been overlooked,' instancing a 'suppurating mesenteric gland,' a 'gumboil,' or a 'whitlow.' An examination of the seventy-six cases tabulated by them shows that in seven out of the ten cases in which the cause is not obvious the abscesses were encysted; in one case a second non-encysted abscess also was found; in two the abscesses were distinctly stated not to have been encysted; and in one, its condition is not mentioned. In the sixty-six cases in which the abscesses were obviously secondary to other lesions, only seven are noted as encysted; more of course may have been so, without the recorders of the cases noticing it; but, in nearly half, the abscesses are either distinctly stated not to have been encysted, or the accompanying description makes it highly improbable that they were. Though many undoubtedly traumatic abscesses of the brain become encysted, I think the much greater frequency with which cysts are found in cases where no cause is obvious is an important fact, and should make us hesitate to refer them to 'a fall' or blow, unless some evidence of injury or concussion at the time of the accident is present. The objection that encysted abscesses are chronic does not appear to me valid, for from their very chronicity we are unable to ascertain all the facts bearing on the case, and are all the more apt to refer them to a wrong cause. In the case before us there was no cause discovered in the post-mortem room, but a history of a 'fall' into water and great mental excitement existed, and from that time the acuter symptoms appear to date, whilst the

* Vol. ii. p. 552 et seq.

history of impaired health reached back for many months. In two of Dr. Abercrombie's* three cases of encysted abscess no cause is apparent; in the third it was associated with abscesses in the lungs. In one of his cases (XLI.), the eyes were in perpetual motion, a symptom that was very marked in Case XIII. of cerebellar abscess consequent on disease of the ear. The almost total absence of pain in this case appears to me noteworthy. When admitted the leading symptoms were languor, fretfulness, and occasional vomiting.

The two following cases are of unusual interest, presenting pathological appearances at first sight very similar, though dependent, I believe, on widely dissimilar causes.

CASE XVI.—*Acute meningitis—Headache and vomiting—Delirium—Coma—Death on the third day.*

Edward M., æt. 18; admitted July 1; died July 1, 1867.

History.—A scrofulous-looking lad, by occupation a newspaper boy. He was taken ill on June 29, about the middle of the day, with severe pain in the head and vomiting. The headache and vomiting continued until the middle of the next day, when he became delirious. When admitted he was in a state of violent delirium, so that it was difficult to keep him in bed. His pupils were dilated; pulse 72, irregular. The skin did not feel hot; tongue furred, moist; bowels not open for three days, and no urine had been passed for thirty hours. Soon after admission two pints of urine of low specific gravity, loaded with albumen and containing some blood, were drawn off. A few hours after admission he became comatose, and died the same evening. It was subsequently ascertained that he had had œdema of the feet and legs some twelve months previously, and that he was subject to attacks of epistaxis, which were occasionally very severe.

Autopsy thirteen hours after death.—Body fairly nourished. Rigor mortis considerable. Scalp congested and adherent to the bones of the skull. Calvaria natural. The longitudinal sinus empty. On reflecting the dura mater the surfaces of the hemispheres were found covered with pus and the convolutions were slightly flattened. In the longitudinal fissure pretty firmly organised lymph was met with; at the base of the brain there was a considerable amount of pus, but little lymph. The sides of the Sylvian fissures, except just at

* Loc. cit. p. 99.

their commencement, were not unduly matted together, and the vessels and membrane in them appeared natural. The ventricles contained about the usual quantity of perfectly clear serous fluid; the anterior horns were of unusual size, whilst the posterior and the hippocampus minor were small. The brain substance was soft and the puncta cruenta unusually numerous; the softening was most marked in the temporal lobes.

Both pleural cavities contained numerous firm adhesions; the lungs were œdematous and congested, but no tubercle was found in them or in the bronchial glands.

The aortic valves were thickened and shortened, and had a few warty granulations on them. The mitral valve was slightly affected in a similar manner. Weight of the heart ℥xij. The spleen soft and pulpy and firmly adherent to the contiguous parts. Liver congested; in other respects natural.

The capsules of the kidneys were adherent, splitting; the organs themselves very congested; the stellate veins being unusually marked, but on section their tissue appeared healthy to the naked eye. The weight of the right ℥v¾, of the left ℥vij.

CASE XVII.—*Puriform lymph at the base of the brain, and on the cerebellum—Necrosis of the ribs—Rheumatic (?) pains—Pain in the head—Impairment of respiratory action—Death without any convulsions or paralysis.*

William F., æt. 43; admitted March 24; died April 8, 1868.

History.—Eleven weeks before admission was supposed to have had acute rheumatism. On admission he complained of severe shooting and stabbing pain in the left side, which simulated pleurisy, and was much increased on cough; also of pain in the head, which prevented his sleeping at night. He remained much in the same condition until a few hours before death, when his breathing became laboured and difficult, and he gradually sank without any convulsion, paralysis, or delirium.

Autopsy twenty-six hours after death.—Body poorly nourished. Rigor mortis well marked. In reflecting the muscles of the thorax two small abscesses were found; the upper in connection with the junction of the fourth costal cartilage and rib. The lower at the junction of the ninth costal cartilage and rib. The end of the fourth rib was necrosed, as also

that of the ninth throughout the greater part of its length, and surrounded by fresh bony material. Both the pulmonary and parietal pleuræ in close connection with the necrosed ribs were perfectly healthy. The lungs were emphysematous. The heart, liver, and spleen were natural. The capsule of the left kidney was slightly adherent; on its posterior and upper surface a mass of pale fawn colour projected, which proved to be an abscess the size of a hazel nut; near it was a moderate sized infarctus. The kidney was greatly congested and weighed ℥ vij. The right was congested; in other respects natural. The scalp separated naturally. The dura mater appeared thicker than usual and had a somewhat cartilaginous feel. On reflecting the dura mater the surface of the arachnoid was rather opaque, and there was a large quantity of subarachnoid fluid. At the base of the brain a mass of greenish coloured puriform lymph was found covering the right side of the pons, and about half of the medulla oblongata was covered with similar lymph; it was spread also over the under surface of the cerebellum, dipping down into the groove round the flocculus, but had nowhere invaded the brain substance. The whole patch of lymph was of an irregular oval shape about 1·5 inches in length by the same in its greatest breadth; through the midst of this lymph the right inferior cerebellar artery passed, and its canal was occupied by a firm fibrinous clot of some standing. A portion of the petrous bone in advance of the internal auditory meatus was slightly discoloured, but the dura mater over it was adherent and natural, and there was no discharge from the ear, nor, as far as I could learn, had there ever been during life. Case XVI. appears to be one of acute arachnitis, similar to that not unfrequently met with after injury to the scalp or skull. No evidence of any concussion or blow was obtained either from the friends, or from a careful investigation of the body itself. The presence of albumen, and the low specific gravity of the urine drawn off, might possibly have been owing to the brain disturbance; but the history of œdema of the feet and legs some twelve months previously, and the condition of the kidneys, which were large and had their capsules unduly adherent,* with the stellate veins prominent, render it probable that the brain affection was coexistent with, if not dependent on, uræmic poisoning. Case XVII. I regard as one of pyæmia, as is shown by the presence of an abscess in the

* I have unfortunately mislaid or lost the note of the microscopical appearances of the kidneys, and do not venture to trust to my unassisted recollection.

kidney; the cerebral mischief had as its starting-point the occluded inferior cerebellar artery. The latency of the brain affection, with so much mischief present in the meninges of the brain, is remarkable. The arachnitis appeared to be nearly as acute as in Case XVI., yet there was no delirium, and the pain in the head appears to have been almost as severe on admission as during any of the remainder of his life. In Case XVII. we meet with a chronic disease which was tolerated by the brain, and led to no acute disturbance of its functions; whilst in Case XVI. the whole disease appears to have run its course in three days with violent functional disturbance.

CASE XVIII.—*Embolism of the left middle cerebral artery—Facial paralysis—Imperfect speech—Hemiplegia—Death in five months.*

Charles M., æt. 45; admitted March 7; died August 11, 1867.

History.—When admitted he was in an excited state, and was unable to speak; there was partial facial palsy. After being in the Hospital some time hemiplegia of the right side came on, and the imperfection of speech remained and became worse; he could only articulate two or three words distinctly—such as 'probable,' 'damn,' 'yes'—and was unable to put out his tongue. He appeared quite sensible until near the close of his life, and made frequent efforts to speak, which always eventuated in one or other of the above words. The hemiplegia varied a little from time to time, but latterly was quite complete. He gradually became feebler, and died without any fresh brain symptoms.

Autopsy twenty-eight hours after death.—Body emaciated; decomposition advancing; rigor mortis inconsiderable. The scalp was very adherent to the bones of the head, and posteriorly œdematous; calvaria separated naturally. On reflecting the dura mater the surface of the right hemisphere was natural, but very pale. The left hemisphere was very soft, feeling quite diffluent, but the convolutions retained their distinctness. On section of the left hemisphere the greater part of it was found to be of the consistency of thin cream, and of a dirty stone colour. The convolutions near the vertex and frontal lobe were not so much softened as those of the temporal lobe. The fluid in the left ventricle was clear, and its walls natural. The sensory ganglia on the left side were softer than

those on the right, the right half of the brain being pallid and anæmic, but firm.

The arteries at the base of the brain were healthy, but the main trunk of the left middle cerebral artery, as well as its principal divisions, was occupied by an organised clot, adherent at points only to the walls of the vessels, and by no means obliterating the whole canal.

There were a few thread-like pericardial adhesions over the left ventricle. The right chambers of the heart were somewhat dilated and hypertrophied, the left very much so. The aortic valves were rendered rigid by calcareous deposit within them, and the cusp next the pulmonary artery had given way, being turned downwards towards the ventricle. The muscular substance of the heart was very soft and mottled with pale patches. The heart weighed ℥xxv.ss. The aorta was healthy; embolic masses were found in the kidneys but not in the spleen; the liver was nutmeggy.

The pathological changes recorded speak so eloquently for themselves, that remarks on them are almost superfluous. I apprehend that the artery was at first wholly occluded, and that the nutrition of the brain became so impaired that when the canal of the vessel again became pervious reparation of the brain substance was impossible. I am unable to suggest any explanation why the clot should have become organised and adherent to the sides of the artery at distant points only, instead of leading to its obliteration and conversion into a fibrous cord, as more usually occurs.

CASE XIX.—*Carcinomatous tumours in the brain—Apoplectic seizure—Partial hemiplegia—Second attack—Unilateral convulsions—Coma—Death in twenty-eight hours.*

George P., æt. 49; admitted January 20; died January 22, 1868.

History.—He was picked up insensible by the police, and brought to the Hospital. Six weeks previously he had been found lying at the bottom of some steps near his abode; when found he was conscious, but did not know what had happened; since that time the left side had been partially paralysed, but not sufficiently so to prevent his getting about. He had lately passed large quantities of pale urine, and his appetite had been ravenous.

When brought to the Hospital he was insensible, his pupils natural, but insensible to light. 'He looked like a man

recovering from an epileptic fit.' The left side appeared to be paralysed.

By 5 P.M., two hours and a half after admission, he had had nine attacks of convulsions; they were unilateral, affecting the left side. Pulse 130, full. Cupped to six ounces, after which he perspired freely and the pulse became softer. 10 P.M.—Has had a constant succession of fits, each lasting about three minutes, and there being about a minute's interval between them. Bowels have acted, and he has passed a small quantity of urine loaded with lithates, and containing a large quantity of albumen.

January 21.—No fits since 10.30 P.M. last night; coma is becoming deeper, and breathing stertorous; died at 3.30 P.M.

Autopsy twenty-one hours after death.—Body fairly nourished, much internal fat; rigor mortis considerable; scalp rather adherent; calvaria somewhat congested. On reflecting the dura mater the convolutions were seen slightly flattened; the sides of the longitudinal fissure were adherent. The ventricles were distended with clear serum, and their walls firm and tough. The substance of the hemispheres was rather congested and watery, but firm and even tough. Situated in the right crus of the cerebrum, below the corpora quadrigemina and posterior portion of the optic thalamus, was found a rounded, soft, pink-coloured mass, about the size of a small marble, looking at first sight not unlike a cyst; round about this mass was a small quantity of extravasated blood; but the tumour itself was of a delicate pinkish-grey colour, very similar to that of the grey matter of the brain when congested. The brain in its immediate vicinity was not altered in structure or softened. Situated in the anterior portion of the left optic thalamus just anterior to the soft commissure, and slightly encroaching on the corpus striatum, was a similar but rather larger mass, and round this there was no extravasation of blood; over its surface two or three distended vessels could be distinctly traced. By its pressure it had made a small pit on the inner side of the right optic thalamus, but was not continuous with the substance of the right optic thalamus.

The lungs posteriorly were congested and softened.

The heart was slightly hypertrophied on the left side.

The kidneys were granular and contained numerous small cysts; externally they were loaded with fat. The supra-renal bodies were large and their cortex contained yellow-coloured masses. These when examined under the microscope were

composed entirely of oil globules, and a mere granular and molecular débris. The remaining viscera were healthy.

Portions of the cerebral tumour examined microscopically consisted almost entirely of rounded nucleated cells of a very uniform size, and a little smaller than white blood-globules. Both in the microscopic appearances and to the naked eye the tumours answer most closely to Virchow's description of Glioma of the brain.*

This specimen after being for long in spirit was shown at the Pathological Society, and referred to the committee on morbid growths. In their report they stated, first, that the specimen was not in a fit condition for examination; and, secondly, that they saw no cause to consider the changes which had occurred in the brain tissue to be other than hæmorrhagic effusions. I presume those who examined the specimen, investigated the discoloured portions round the tumour where hæmorrhage had taken place, and not the adventitious growth itself. The drawing taken of the specimen in the recent state, as well as the opinion of all who then saw it, is, I think, sufficient evidence with my own to counterbalance their report. Primary cancer of the brain is a comparatively rare form of carcinoma, and when it occurs, Rokitansky says, 'there is usually but one adventitious growth in the brain, but when there are two it is interesting to notice that they not uncommonly occur symmetrically in corresponding portions of the brain.' Virchow † also states that Glioma of the brain is usually solitary, adding that this is especially true of the soft forms, whilst the hard are often multiple. This instance forms an exception to the general rule, both adventitious growths being situated on the same side of the brain.

The succeeding case is one of syphilitic gumma in the membranes of the brain, originating probably in the dura mater. Dr. Abercrombie ‡ has given a description of a tumour very similar in appearance to the one here described, and Dr. Wilks has both described and figured one nearly identical.§

CASE XX.—*Syphilitic cachexia, with deposits in the dura mater and liver—Epileptiform convulsions—Coma—Death.*

John B., æt. 34; admitted June 6; died July 11, 1867.

History.—A sailor, broken down by syphilis and tropical

* 'Die krankhaften Geschwülste,' 2ter Band, p. 131, 137.
† Loc. cit. p. 147. ‡ Loc. cit. Case vi. p. 45.
§ 'Guy's Hospital Reports,' 1866, p. 49.

fever, from both of which he had frequently suffered. When admitted he had indistinct symptoms of ague, with pains in all his limbs, enlargement of the clavicles and nodes on the tibiæ, but made no complaint of his head; at first he improved, but was suddenly seized the day before his death with epileptiform convulsions, and became comatose and died.

Autopsy forty hours after death.—Body poorly nourished; the skin over the face, arms, and legs of dingy brown colour; scars of numerous circular ulcers about the legs and ankles, the scar of a bubo in the right groin, and nodes on the clavicles and tibiæ. Rigor mortis had nearly passed off, and there was commencing green discoloration of the abdomen. The bones of the skull were extremely thick and dense, especially the frontal bone; the dura mater natural except over the anterior surface of the right frontal lobe, where a tumour the size of a walnut was found in connection with the membrane; around the tumour all the membranes were adherent, and it appeared to have involved them all. The tumour had caused partial absorption of the frontal bone, which was somewhat altered in texture, the diploe being white and having a granular appearance, and though thick was not hard; the portion which was most altered in appearance was surrounded by a ring of increased vascularity. On section the tumour was of a bluish-white colour and very firm; its limits, where it came in contact with the cerebral hemisphere, were irregular but well defined, the brain substance in immediate connection with it being diffluent. Throughout the cerebral hemispheres the puncta cruenta were unusually numerous and large, and the brain was soft and œdematous. The mass, when examined microscopically, presented an indistinctly corpusculated appearance, here and there fibrillated, but without any distinct cells or fibres, and chiefly made up of granular and molecular elements. No other adventitious masses were found in the brain or its meninges.

The pericardium contained ʒj. of slightly turbid fluid. The left pleural cavity contained a little similar fluid and some loose lymph. The lower lobe of the left lung was in a state of hypostatic pneumonia. The right pleura and lung were natural. The bronchial glands œdematous. Numerous firm membraniform bridles passed between the upper surface of the liver and the diaphragm; the surface of the liver was irregular, each bridle springing from a depressed portion, and in the centre of each depression a small mass similar in colour and general appearance to the mass in the brain was found, situated imme-

diately beneath the liver capsule. These masses, examined microscopically, furnished the same granular and indistinctly corpusculated materials as the larger tumour in the brain. The liver tissue generally appeared healthy; it weighed 3lbs. 8½oz. The gall bladder contained thick, dark olive-green bile. The remaining viscera and the testes were normal.

CASE XXI.—*Tubercular meningitis—No tubercle detected in the thoracic or abdominal viscera—Diarrhœa, pain in the head and vomiting—Constipation—Death on the twenty-second or twenty-third day.*

Alice C., æt. 6; admitted July 23; died August 4, 1867.

History.—Her father brought her to my out-patient room on the 19th. She had then been ill five days. Her illness commenced with pain in the head and diarrhœa; this was followed by vomiting, and since the accession of the vomiting the bowels had been confined. Her father refused to leave her in the Hospital, but after four days she was brought back and admitted. On the 24th she had no action of the bowels and no vomiting; had passed a quiet night, and appeared heavy and drowsy; pupils dilated and acting sluggishly; abdomen retracted; temperature 100·6° F.; pulse 76, regular. She remained in much the same state until the evening of the 29th, when she was very restless. On the 31st some slight convulsive twitchings of the facial muscles were noted. August 2.—Pupils dilated; the conjunctivæ and the skin appeared wholly deprived of sensation. Temp. 99·7° F.; pulse 112, irregular; respirations 20. August 3.—The left arm was in a state of tonic spasm; temp. 101° F.; respirations 32.

Autopsy about twenty-four hours after death.—Body fairly nourished; green discoloration of the abdomen. The scalp and calvaria natural; the pia mater was somewhat congested, and numerous small white specks were visible along the course of the vessels. At the base of the brain an abundance of ordinary tubercular lymph was found. The Sylvian fissures had their sides unduly adherent. The convolutions were flattened and the ventricles were distended with clear fluid. The fornix and velum interpositum were softened. The cerebellum was much paler than the rest of the brain, which was nowhere much congested.

No tubercle was detected in any of the other organs.

The next case is a strong contrast to the above, general

tuberculosis of the viscera having occurred. Though no tubercle was detected by the naked eye in or on the brain, the softened condition of that organ, and the diffusion of tubercle through the other viscera, render it highly improbable that it had escaped.

CASE XXII.—*Tubercular meningitis (?)—Tuberculosis of thoracic and abdominal viscera—Slight cough—Diarrhœa—Convulsions—Death about twenty hours after the first convulsion.*

Annie B., æt. 2½; admitted May 11; died May 23, 1868.

History.—Admitted suffering from slight cough and severe diarrhœa. The child continued much in the same condition until the evening of the 22nd, when it was seized with convulsions, and continued to have a series of convulsive attacks until noon of the 23rd, when it died convulsed. The child had always been sickly.

Autopsy forty-eight hours after death.—Body poorly nourished; rigor mortis nearly passed off. Scalp and calvaria natural. The surface of the brain a good deal congested. The brain was soft and watery, and the gray matter had a pinkish tinge, but no granulations on or thickening of the membranes* were detected with the naked eye.

On both layers of the pleuræ were situated small greyish-white granulations the size of split hemp-seeds. The pleuræ were universally adherent. The volume of the lungs was increased, they felt solid, and were stuffed with tubercle. The trachea and bronchi pallid. The mucous membrane of the glottis looked succulent, and was thrown into large longitudinal folds, but it could not be said to be œdematous.

The bronchial glands were infiltrated by firm yellow tubercle.

The spleen, liver, and kidneys, were all filled with small masses of yellowish white tubercle, and the peritonæal capsules of them, and of the gall-bladder, covered with small granulations.

The mesenteric glands were enlarged, and occupied by firm yellow tubercle. The mucous membrane of the intestine was the seat of tubercle which had here and there begun to ulcerate.

The peritonæum lining the pelvis was free from granulations.

* Unfortunately, the portions of membranes reserved for microscopical examination were spoilt before I had an opportunity of examining them.

CASE XXIII.—*Tubercular meningitis—Tubercle on the peritonæum, and in the substance of the liver, spleen, and bronchial glands.*

William M., æt. 8; admitted July 10; died July 14, 1868.

No history was obtained.

The convolutions of the hemispheres flattened. A large quantity of slightly turbid fluid in the ventricles of the brain, which were much dilated. The fornix and velum interpositum much softened; the whole brain watery. At the base of the brain a small quantity of yellowish lymph was met with.

There were a few small masses of tubercle on the pulmonary pleura.

The bronchial glands contained tubercle, as did the liver and spleen.

The peritonæum over the under surface of the diaphragm was studded with small tubercular masses. The lungs contained no tubercle.

CASE XXIV.—*Tubercular meningitis—No tubercle noted in any other organ.*

Charles B., æt. 5; admitted June 20; died July 4, 1868.

History.—His mother attributed his illness to a kick he had received over the eye three weeks before admission.

No note of his symptoms was taken.

Autopsy twenty-eight hours after death.—On the surface of the brain, especially over the left hemisphere, were small greyish granular deposits about the size of pins' heads, lying along the course of the vessels. At the base of the brain an abundant thick deposition of yellowish lymph completely covering the origins of the cranial nerves was found. The ventricles contained a considerable quantity of fluid. The posterior portion of the brain much softened, and the fornix and contiguous parts almost diffluent.

No tubercle was noted as present in other organs.

CASE XXV.—*Tubercular meningitis—Tubercle in the bronchial glands—History of an injury to the head—Vomiting, constipation, paralysis, and anæsthesia of right side; jactitation of left—Coma—Death nineteen or twenty days from commencement of symptoms.*

Stephen M., æt. 10; admitted March 7; died March 16, 1868.

History.—He had always enjoyed good health, and had worked as a bricklayer's lad. Ten days before admission the man with whom he worked was stated to have knocked his head against the wall, and some of the other workmen to have done the same the next day;* two days afterwards he vomited, and the vomiting has remained constant ever since. The day before admission he had a fit, with loss of consciousness and speech. On admission his face was flushed, and there was facial paralysis on the right side; the right arm and leg were also paralysed, and there appeared to be complete anæsthesia of the skin over them. The left arm and leg were continually tossed about, so that it was impossible to keep any bed-clothes on him. He moaned frequently, but was quite unconscious. Pulse 112, moderate volume. The bowels are reported not to have acted for twelve days. He passed urine freely in the bed.

On the 8th he appeared slightly more conscious, but did not speak. The bowels had acted freely after an enema. No vomiting. On the 9th he had regained power slightly on the right side. During the next four days he remained much in the same condition (the bowels acting after castor oil), and taking food pretty readily. He scarcely ever spoke, but moaned and cried out a good deal at times. On the 14th the stupor had increased, and he did not take food so willingly. On the 15th he became quite comatose, and died the next day without recovering sensibility.

Autopsy twenty-nine hours after death.—No trace of injury to the soft parts or to the bones of the skull was perceptible. The body had a considerable amount of subcutaneous fat. The calvaria natural. Dura mater congested. The convolutions were flattened, and the vessels of the pia mater full of blood; along the course of the vessels over the temporal lobes numerous small white specks were found. At the base of the optic commissure, the interpeduncular space and the pons were

* At the inquest it appeared doubtful whether any violence at all had been used: the father and mother of the deceased had evidently an old grudge against the accused. Evidence was given to show that the deceased made no complaint of being hurt, and his parents admitted that there was no bruise of the head.

almost hidden by greenish lymph, which spread also into the Sylvian fissures.

The bronchial glands were occupied by crude grey tubercle. At the apex of the left lung a small hard body the size of a pin's head was met with, but with these exceptions no trace of anything resembling tubercle was found.

All the abdominal organs and the peritonæum appeared healthy.

CASE XXVI.—*Tubercular meningitis—Tubercle in the lungs —Slight rigors—Vomiting—Pain in the head very slight— Death on the fifteenth day.*

Henry P., æt. 23; admitted May 18; died May 26, 1868.

History.—Applied as an out-patient on May 14; stated that three days previously, after exposure to wet, he had had slight shivering, followed by sickness. On the 18th he returned appearing much worse, the vomiting not having ceased, but not complaining of pain anywhere. He was then admitted. The vomiting continued for the next twenty-four hours; tongue clean, bowels acted, pulse rapid. Slight pain in the head, and the pupils unequal. On the evening of the 19th the vomiting ceased. During the rest of his life in the Hospital he was sleepless; wandered slightly at times, especially at night, but was not noisy; made scarcely any complaint of pain in the head; his pupils remained unequal and his pulse rapid; his breathing gradually became laboured and oppressed, and he died without the occurrence of any convulsions on the 26th. His temperature never exceeded 100° F.

Autopsy twenty-four hours after death.—Body well nourished; rigor mortis marked. There was an old scar on the right superciliary ridge, but it was confined to the skin, not being adherent to the pericranium, and the frontal bone beneath the scar being quite natural. On removing the calvaria and reflecting the dura mater, flattening of the cerebral convolutions was very marked. There were numerous small tubercles found in the membranes at the base of the brain, and a smaller number on the superior portion of the membranes.

The ventricles contained a considerable quantity of turbid fluid, and the surfaces of the lateral ventricles were covered with small, elevated, sand-like bodies. The central portions of the cerebrum and the fornix were much softened.

The lungs were very thickly studded with miliary tubercles.

The rest of the viscera, as far as they were seen, were healthy.

CASE XXVII.— *Tubercular meningitis—Masses of tubercle in the cerebellum—Tubercle of the serous membranes, lungs, and mucous membrane of the bladder and intestines—Feeble health, frequent pain in the head, occasional vomiting, and strabismus—No convulsions or coma.*

John William H., æt. 7½; admitted February 20; died March 17, 1868.

History.—The child was stated to have been in bad health for a year, and to have complained frequently of pain in the head for five months. A fortnight before admission strabismus was observed, but it did not remain constant. He vomited occasionally, and complained of giddiness. During his life in the Hospital he had no vomiting, the bowels were easily acted on, and the motions copious. He had occasional strabismus, but made very little complaint of pain in the head. His pulse was regular and very rapid. He complained chiefly of pain in the belly, and died apparently from exhaustion.

Autopsy forty-eight hours after death.—Body fairly nourished; the subcutaneous tissue did not contain much fat, but the sub-peritonæal tissue was in places loaded with fat; rigor mortis considerable. Scalp unduly adhered to the pericranium. The skull measured 19½ inches in its largest circumference. Calvaria natural. The arachnoid was dry and sticky, and the convolutions of the hemispheres slightly flattened. The membranes at the base of the brain, from the optic commissure to the pons were dotted with small white specks, but no lymph was found on them; they were thickened and much matted together in the fissures of Sylvius. On slicing the cerebral hemispheres the brain was found firm and pallid. The lateral ventricles greatly dilated. The septum lucidum broken down, and the fornix much softened. The cerebellum was infiltrated with firm yellow tubercle, which dipped in between the convolutions, and two or three distinct masses, the size of small beans, were found in the left cerebellar hemisphere. The portions of the cerebellum in immediate connection with these masses were softened and nearly diffluent. The pleuræ were covered with small granulations more easily felt than seen. The lungs contained a quantity of grey miliary tubercle. The bronchial glands contained grey tubercle.

The capsule of the liver was studded with small whitish granulations, from which bands of membrane passed to the under surface of the diaphragm.

The liver itself appeared quite healthy. The spleen was pale and soft, no trace of tubercle being detected in its substance, but numerous bands of adhesions connected its capsule with the surrounding parts. The kidneys appeared quite healthy.

The mucous membrane of the bladder was ulcerated at the fundus over a space the size of a shilling. The peritonæal surface of the bladder was at this point adherent to the rectum, which was also ulcerated, as were the small intestines throughout the greater portion of their course. The mesentery itself appeared free from tubercle, but the serous coat of the intestine was covered thickly with small opaque granulations.

CASE XXVIII.—*Tubercular meningitis—Tubercle on the serous membranes—Gradual loss of power in the lower extremities—Vomiting—Coma—Duration of symptoms uncertain.*

Henry R., æt. 16; admitted July 18; died August 2, 1868.

History.—On admission he appeared to be suffering from extreme debility, and some slight loss of power in the right leg. Two months before admission he was stated to have had fever, was confined to his bed for three weeks, and was delirious during the course of his illness; after recovering sufficiently to leave his bed, a fortnight elapsed before any impairment of power in the leg was noticed, and the loss of power gradually increased up to the time of his admission. Sensation in the limb appeared unaffected. At first he appeared to improve, but on the 24th his pulse was observed to be irregular. On the 25th vomiting occurred, which continued constant, until he became comatose during the night of the 29th. He remained in a comatose condition until a few hours before his death, when he, to a certain extent, recovered his senses, recognising his mother, and taking food.

Autopsy thirty hours after death.—Body greatly emaciated; rigor mortis considerable. Scalp and calvaria natural. The vessels of the pia mater somewhat congested. The sides of the longitudinal fissures, as also those of the fissures of Sylvius, were unduly adherent. A patch of dense yellow tubercular lymph was found situated on the posterior lobe of the left hemisphere; it was most abundant on the inner aspect of the hemisphere, and in the neighbourhood of the internal parieto-occipital fissure. The lymph appeared to be situated beneath the pia mater, and dipped with it into the sulci, section of the

brain disclosing the yellow lymph passing down with the membrane to the bottom of the sulci. The brain substance itself, in immediate connection with the patch of lymph, was slightly congested, but not softened; the rest of the brain and its membranes appeared natural.

The right pleural cavity contained a few soft adhesions, and the pulmonary pleura was extensively ecchymosed, and here and there had a little lymph upon it. The lower lobe of the right lung was congested and solidified, containing scarcely any air; the upper lobe had a few hard nodules of grey tubercle scattered throughout it. The left lung was uniformly infiltrated with masses of tubercle. The bronchial glands were enlarged, and occupied by yellow tubercle. The spleen was adherent to the diaphragm and the abdominal wall, and contained numerous masses of yellow tubercle of the size of peas.

The capsule of the liver was everywhere studded with small opaque granulations, and numerous adhesions passed from its upper surface to the diaphragm. No deposit could be detected in the substance of the liver itself.

Scattered over the surface of the kidneys were numerous small whitish specks, and similar ones were found in the cortical substance on section of the organs; the spleen contained numerous small yellow rounded masses of tubercle. The peritonæum covering the stomach and abdominal walls was everywhere covered with small flattened granulations, varying in size from that of a split pea to an almost invisible speck; the mesentery and pelvic part of the peritonæum were quite free from granulations.

CASE XXIX.—*Tubercular meningitis—Tubercle and cavities in the lungs—Phthisis—Partial paralysis—Noisy delirium—Death—Advent of cerebral symptoms uncertain.*

Mary Ann G., æt. 47; admitted January 31; died February 13, 1868.

History.—A poorly nourished woman, who, when admitted, appeared to be in the last stage of phthisis. She was extremely exhausted, her body slightly anasarcous, and considerable anasarca of the lower limbs. She was apparently densely stupid, but answered questions coherently. At first she seemed to improve a little and be a little stronger, but after being a week in hospital, she partly lost control over the sphincters, and her lower extremities became almost wholly paralysed. She now had considerable delirium at night, sing-

ing and shouting, but was tolerably quiet during the day, but sleepless. If vomiting occurred at all it was not persistent or marked. She sank at last gradually.

Autopsy thirty hours after death.—Body poorly nourished and anasarcous; anasarca most marked in the right lower extremity. The cerebral convolutions prominent, showing no evidence of flattening. Much serosity in the subarachnoid spaces. In the Sylvian fissures, and at the base of the brain, numerous small granules and specks of tubercle were scattered throughout the pia mater, and the whole membrane was opaque. The brain appeared atrophied. There was no tubercle in the brain substance itself.

There were numerous firm and tough adhesions in the left pleural cavity, most abundant near the apex; some also in the right, but neither so numerous nor so tough. Both lungs were stuffed with tubercles, and at the apex of the left was a large irregular cavity. The lungs generally were congested. No tubercle was noticed as present in the other organs.

CASE XXX.—*Tubercular meningitis—Tubercle on the serous membranes, and in the mucous membrane of the large intestine—Pain in the head—Vomiting—Coma—Death on the twelfth day from accession of cerebral symptoms.*

Mary Ann S., æt. 24; admitted October 15; died October 17, 1867.

History.—During her confinement, twelve months before the commencement of her illness, she had convulsions; suckled her child up to October 10, four days after the commencement of her present illness, which began with severe pain in her head and vomiting on October 6. On the 7th headache was worse and the light painful to her eyes. On the 9th she first became delirious. On the 10th she could with difficulty be roused; the bowels relaxed, the stools reported to be light in colour and watery. On the 13th she was quite sensible again. Soon after admission on the 15th she became comatose, and died without again recovering her senses.

Autopsy nine and a half hours after death.—Body fairly nourished; still warm. Rigor mortis present and considerable.

The scalp and calvaria natural. The dura mater a little congested; the convolutions of the cerebral hemispheres appeared slightly flattened, and a few transparent granulations could be seen situated along the course of the vessels; similar granulations were found on the parietal layer of the arachnoid.

At the base of the brain a good deal of serum was present, and some membraniform lymph about the circle of Willis; the Sylvian fissures also were glued together by recent lymph. The fluid in the ventricles was clear, and not more abundant than usual. The septum lucidum and fornix were soft and almost diffluent, and the temporal lobes were much softer than the rest of the cerebrum. The sensory ganglia were also softer than usual, but did not appear congested or altered in structure.

Pleuræ. The left was studded with small transparent granulations; at the base of the lung it was ecchymosed, and covered with a little recent lymph. The cavity of the right pleura was obliterated, but the layers could he separated easily, and the granulations on the pleura were larger than those on the left. At the apex of the right lung a small calcareous mass was found, and a large quantity of miliary tubercle was scattered throughout both lungs. Bronchial glands contained cheesy tubercle.

Pericardium and heart natural.

The capsule of the liver looked as if sanded over with small transparent granulations. On section the organ was pale and soft. No tubercle detectable in its tissue. Weight 2 lbs. 14 oz. The capsule of the spleen was roughened by adherent lymph, but no tubercle could be discovered in it. The capsules of the kidneys separated easily, leaving the surface smooth, but irregularly congested; in their substance numerous small white specks were seen on section. Scattered throughout the colon were numerous small tubercular ulcers. Attached to the right broad ligament were a few small cysts. The mammæ contained a little thin opalescent fluid.

The microscopical appearances presented by the lymph from the Sylvian fissures, the granulations on the peritonæum, and the masses in the lungs, were almost identical. In the lymph from the brain the corpuscular elements were embedded in a larger quantity of fibrillated material.

CASE XXXI.—*Tubercular meningitis and pericarditis—Death on the seventeenth day from advent of cerebral symptoms.*

Mary Ann Y., æt. 24; admitted June 20; died June 26, 1867.

History.—A cook. She had not been in the enjoyment of very good health for a year or more, and on June 10 she awoke with a severe headache, and in the course of the day had a fit, remaining in a state of insensibility for nearly three hours;

she was able to continue her work, although feeling ill and frequently vomiting, until the 16th, when she became worse, and was obliged to take to her bed, suffering very severely from headache. During her life in the Hospital, she appeared stupid and heavy, but answered questions rationally, and took nourishment when offered her, but never asked for anything. At night there was always an increase of the cerebral symptoms, and occasionally she screamed violently. Her pupils were natural, but acted sluggishly, and strabismus was noticed once or twice. On the day of her death she appeared brighter and more conscious, and complained of severe pain in the back of the head. Her urine and motions were passed unconsciously, the bowels being freely acted on by Hst. magn. sulph. ℥i. ter die. She had no vomiting whilst in the Hospital. The pulse was rapid, 120 to 130, and the temperature of the skin was constant at 100° to 101° F.

Autopsy thirteen hours after death.—Rigor mortis slight. A large quantity of subcutaneous fat was present, especially in the abdominal parietes. The scalp and calvaria natural. In the longitudinal sinus was a small recent clot. The vessels of the pia mater were uniformly congested, and along the course of some of them the membrane was studded with small white specks. About the optic commissure there was an abundant deposition of lymph, as in the fissures of Sylvius, where the lymph felt hard and gritty. On section the brain substance was firm. The ventricles contained no undue quantity of serosity. The choroid plexus and velum interpositum appeared thickened by the presence of œdematous lymph. The under surface and pillars of the fornix were softened.

No adhesions were met with in either pleura. At the apex of the right lung, a small hard greyish-white mass the size of a pea was found, and several similar masses in the upper lobe of the left lung. Some of the bronchial glands were enlarged, containing cheesy material of a yellowish-grey colour. The two layers of the pericardium were universally adherent, and when separated small masses of a grey colour were found on both the visceral and parietal layers; these were most abundant over the posterior surface of the left ventricle. The grey masses dipped down from the pericardium into the muscular tissue of the heart to the depth of one or two lines. In all other respects the heart was natural, and its weight ℥vij.

The rest of the viscera were natural, no trace of tubercle being detected on the peritonæum, or in the substance of the liver, spleen, or kidneys.

CASE XXXII.—*Tubercular meningitis—Tubercles on the pleuræ and in the lungs and bronchial glands—Headache—Delirium—Coma.*

Ann D., æt. 43; admitted June 23; died July 1, 1868.

History.—Taken ill suddenly a week or more before admission, whilst she was exposed to the sun in a garden. She seemed faint, and her illness was supposed to be due to the heat of the weather. On admission she had the appearance of being very obstinate; she was not at all unconscious, but appeared absorbed, as it were, with her complaint; and it was with difficulty ascertained that she had diffused pain over the head. The night after admission she was very delirious; the headache continued, and her temperature was high. In a day or two inequality of the pupils ensued; she gradually became unconscious, and died without any paralysis of her face or limbs being noticed.

Autopsy twenty-four hours after death.—Body fairly nourished, there being a considerable quantity of subcutaneous fat, and rather an undue quantity about the viscera. Rigor mortis moderate. Scalp natural; the calvaria was of remarkable thickness and density, and was greatly congested, as was the dura mater; on reflecting the latter the arachnoid was found sticky and dry, and the vessels of the pia mater intensely congested. The convolutions appeared slightly flattened, and along the course of the vessels, especially over the lateral region of the left hemisphere, numerous small irregularly shaped masses of a whitish-yellow colour were scattered.

On slicing away the hemispheres, the ventricles were found considerably dilated, and filled with slightly turbid fluid. The septum lucidum was broken down, and the fornix much softened. The choroid plexus was natural, but there was a quantity of adventitious and very œdematous lymph about the velum. The left hippocampus minor and the adjacent brain were very soft, and there were numerous small punctiform extravasations into its substance.

The membranes at the base of the brain and about the optic commissure were matted together by recent lymph. On the superior surface of the cerebellum the membrane was covered with lymph, and on its right hemisphere, above the flocculus, was a patch of lymph half an inch long by a quarter broad, and about one-eighth of an inch in thickness. Scattered over both pleuræ in nearly equal quantity were vast numbers of small whitish granulations, and the lungs were stuffed with grey

miliary tubercle. The bronchial glands contained old cretaceous masses. The peritonæum over the liver had granulations, similar to those on the pleuræ, on it. No tubercle was discovered in the substance of the liver or spleen or any abdominal organ.

CASE XXXIII.—*Tubercular meningitis—Tubercle in the lungs and kidneys—Headache for several months—Intense pain in the head—Vomiting—Coma—Death on the twenty-second day from advent of severe cerebral symptoms.*

John Michael C., æt. 38; admitted November 6; died November 9, 1867.

History.—Was a draper's assistant, and till within ten months of his death had been very intemperate; since leaving off drink had become a great smoker. During the winter of 1866 he had pleurisy, attended with considerable delirium at night. Throughout the summer of 1867 he had complained of headache; on the night of October 20 the headache became intolerable, and he vomited; the headache continued so severe that he was confined to his room, the pain being situated between the eyes and across the forehead. On the 31st he vomited two or three times every hour, and his bowels were very constipated. When admitted he was perfectly insensible, pupils dilated, skin cold and moist, the temperature 98° F., pulse 72, regular but very feeble. During the night of the 6th he was very restless and noisy; got out of bed and knocked the nurse down. He became comatose the following day, and died without recovering sensibility.

Autopsy forty-eight hours after death. Body fairly nourished; rigor mortis great. The scalp was unduly adherent to the bones of the skull. On reflecting the dura mater the pia mater was seen greatly congested, and the convolutions of the hemispheres appeared somewhat flattened. No abnormal appearances were met with, except in the Sylvian fissures where the membranes were matted together. The brain was firm but watery.

Pleuræ. The left was natural, the right universally adherent, and had undergone cartilaginous thickening. The right lung was in a state of partial collapse; near its apex some cicatrix tissue was found, in which was embedded a cretaceous nodule, and throughout the upper lobe were a few masses of miliary tubercle. The left lung was emphysematous and scattered throughout with miliary tubercles.

The capsules of the kidneys separated naturally, leaving the surfaces smooth, but marked by stellate veins. On section the kidney structure appeared nearly natural, but was specked here and there by small white rounded bodies.

In the cæcum one small patch of ulceration (? tubercular) was found. Portions of the pia mater and of the cerebral vessels were examined microscopically. No collection of nuclear elements was found in connection with the walls of the vessels. In the meshes of the pia mater, obtained from the neighbourhood of the Sylvian fissures, vast numbers of nuclear-like elements agglomerated together in groups were discovered; these bodies were very slightly affected by acetic acid, and were undistinguishable from those obtained from the tubercles in the lungs and the white specks in the kidneys.

CASE XXXIV.—*Tubercular meningitis—Tubercle on the pleuræ, and miliary tubercle in the lungs—Feverishness—Extreme delirium and vomiting—Coma—Death about eight or nine days from the commencement of his illness.*

William F., æt. 33; admitted September 27; died September 30, 1867.

History.—Was a member of the fire brigade, and had driven an engine to a fire within a week of his admission; had got very wet, and attributed his illness to that cause. On admission he was very feverish, with a furred tongue inclined to be dry, a quick but not noticeably irregular pulse. His manner was somewhat strange, but his answers, though curt, were clear and succinct. There was some mucous râle in both lungs, but no marked physical signs. During the night he became violently delirious, and could with difficulty be induced to take any food; what he took he vomited. He was violently delirious all the next day, and gradually sank into a state of coma, in which he died.

Autopsy thirty-three hours after death.—Body fairly nourished. Rigor mortis considerable; commencing green discoloration of the abdomen. The scalp and calvaria were natural. The vessels of the dura mater were slightly, and those of the pia mater intensely congested. The convolutions were slightly flattened, and the surface of the brain sticky.

At the base of the brain a quantity of lymph was found effused about the optic commissure and as far back as the pons, and the sides of the Sylvian fissure were unduly adherent,

and matted together by lymph, similar to that found at the base.

The ventricles did not contain an abnormal quantity of fluid.

The white substance of the brain was congested, and the puncta cruenta unusually numerous. No granules of tubercular character were detectable on any of the membranes when examined under the microscope.

The arteries from the base of the brain and Sylvian fissures presented in their smaller branches a beaded appearance; this was due to collections of nuclear-like bodies on their walls. These collections were very numerous, though all were of small size.

The sac of the pericardium was firmly adherent externally to the chest wall; it contained ℥ss. of clear serum. The endocardium was deeply blood-stained.

The two layers of the right pleura were firmly adherent posteriorly and around the apex of the lung. The cavity of the left pleura was wholly obliterated, and the parietal layer was thickened and roughened with small masses of tuberculous material. The lower and part of the upper lobe of the left lung was collapsed, containing no air and sinking in water, its tissue a little softened. The upper third of the upper lobe was stuffed with small grey miliary tubercles of the size of mustard seeds. Similar, but not quite so numerous tubercles were found scattered throughout the upper lobe of the right lung. The trachea and bronchi were in a state of intense congestion. The bronchial glands contained firm tubercle.

No tubercle was found on the peritonæum or in the substance of the viscera. The kidneys were large: their capsules stripped off readily, leaving the surfaces smooth but intensely and irregularly congested. On section the cortical substance appeared unduly increased in amount, and mottled with pale streaks and lines of congestion. The weight of each kidney was ℥vj. Just above the ileo-cæcal valve the mucous membrane of the intestine was ulcerated, but the ulceration had no specific character.

Appended is a tabular view, by which the number of organs affected, and the form in which they were invaded by tubercle, can be seen at a glance.

No.	Name	Age	Brain and Meninges	Pleuræ	Lungs	Liver	Spleen	Peritonæum	Lymphatic Glands	Remarks
21	Alice C. .	6	Granulations along the course of the vessels. Lymph at the base	No tubercle detected	Ditto . .	Ditto . .	Ditto . .	Ditto . .	Ditto . .	.. ..
22	Annie B. .	2½	No tubercle detected by the naked eye	Gray granulations. Universally adherent	Stuffed with tubercle in considerable sized masses	Filled with small masses of yellowish white tubercle	As Liver .	Everywhere except in cavity of pelvis covered with granulations	Bronchial and mesenteric filled with firm yellow tubercle	Kidneys also contained tubercle; and the mucous membrane of intestines
23	William M. .	8	Turbid fluid in ventricles, much softened. A little lymph at base	A few granulations	No tubercle in substance of lungs	Contained tubercle	As Liver .	Upper portion studded with small tubercles	Contained tubercle	.. ..
*24	Charles B. .	5	Granulations on membranes. Abundant lymph at base. Fornix diffluent	.. ..	.. ..	.. ..	.. ..	.. ..	.. ..	No tubercle detected in other organs. Illness ascribed to a kick
25	Stephen M. .	10	Granulations on membranes. Much lymph at base	Natural . .	?	Healthy . .	Healthy . .	Healthy . .	Bronchial occupied by crude grey tubercle	Illness ascribed to violence to the head
*26	Henry P. .	23	Granulations on membranes. Turbid fluid in ventricles. Fornix much softened	Not noted .	Studded with miliary tubercles	.. ..	.. ..	.. ..	.. ..	The rest of the viscera were apparently healthy, but were not examined minutely
27	John William H.	7½	Masses of tubercle in cerebellum. Firm yellow tubercular lymph on the surface of cerebellum. Dilatation of ventricles. Fornix softened	Very small granulations	Miliary tubercle	Capsule covered with granulations	No tubercle .	Covered with granulations	Bronchial contained grey tubercle .	Tubercular ulceration of mucous membrane of bladder and intestines

* The post-mortem investigations of the cases thus marked were not made by myself.

No.	Name	Age	Brain and Meninges	Pleuræ	Lungs	Liver	Spleen	Peritonæum	Lymphatic Glands	Remarks
28	Henry R.	16	Yellow tubercular lymph on left hemisphere	Ecchymosed and smeared with lymph	Left infiltrated with masses of tubercle	Studded over its capsule with granulations. Substance free	Contained numerous masses of yellow tubercle	Studded with granulations, except on mesentery and in the pelvis	Bronchial contained hard yellow tubercle	The surface and cortex of kidneys also studded with white specks of tubercle
*29	Mary Ann G.	47	Much serosity. Tubercle at base and in Sylvian fissures	Numerous firm adhesions	Vomicæ, stuffed with tubercles	.. ..	.. ..	.. ..	.. ..	No tubercle noted in other organs
30	Mary Ann S.	24	Granulations on both parietal and visceral layers. Lymph at the base	Left studded with granulations. Right adherent to lung, granulations larger	Lungs full of miliary tubercles	Capsule studded with granulations. Substance free	Numerous adhesions between capsule and surrounding parts. No tubercle	Except over liver natural	Bronchial glands contained cheesy tubercle	Kidneys contained whitish specks of tubercle. M. membrane of intestines ulcerated
31	Mary Ann Y.	24	Specks along the course of the vessels. Abundant lymph at the base of the brain. Fornix soft	Natural . .	Two or three masses the size of peas in apex of left	Healthy . .	Healthy . .	Healthy . .	Some of the bronchial contained yellowish grey tubercle	Pericardial adhesions with tubercle
32	Anne D. .	43	Granulations on membranes. Slightly turbid fluid in ventricles. Fornix soft. Lymph at base	Thickly studded with granulations	Stuffed with miliary tubercles	Substance healthy	Healthy . .	Granulations over liver and diaphragm	Old cretaceous deposit in bronchial	.. ..
33	John Michael C.	38	Matting together of membranes in Sylvian fissures	Right universal adhesions	Miliary tubercle in both	Healthy . .	Healthy .	Natural . .	.. ..	Patch of ulceration in the cæcum. Kidneys contained numerous white specks
34	William F. .	33	Abundant lymph about optic commissure and pons. No granulations detected	Adherent, and tubercle among the adhesions	Miliary tubercle	Natural . .	Natural . .	Natural . .	Bronchial contained firm tubercle	A patch of ulceration just above the ileo-cæcal valve

* The post-mortem investigation of the case thus marked was not made by myself.

Summary of preceding table:—

The pleuræ were affected by tubercle in	6
The lungs	10
The liver substance	2
„ capsule	6
The spleen	3
Kidneys	4
Peritonæum	6
Mucous membrane of intestines	4
„ „ bladder	1
Bronchial glands	8

In the description of the foregoing autopsies I have used the terms tubercle and tubercular in their widest sense, embracing under the same head not only the grey miliary tubercle of the lung and the granulations of serous membranes, but also the hard yellow exudation met with beneath the membranes of the brain, and in its substance, as well as the dense, hard, grey or yellow interstitial material so common in the bronchial and mesenteric glands. I have done so advisedly, believing that they are all manifestations of a very similar, if not identical, dyscrasia; and that the pathological characters of the adventitious material are modified rather by the structure of the locality in which they occur, than by any essential difference in the originating cause. In the present bewildered condition of the pathology of tubercle, it appears to me advisable to abide a little longer by the nomenclature, if not the theories, of ten or fifteen years ago.

Many more microscopical investigations were made than those recorded in the notes. In every case examined portions of tubercle taken from different organs were compared, and it was generally impossible to distinguish one specimen from another by any definite microscopical characters; this was the case in some instances where the naked eye appearances differed considerably, as in the whitish-yellow bodies in the spleen. The small arterial branches from the pia mater were frequently examined, and in most of the cases of tubercular meningitis, though not in all, collections of nuclear bodies, similar to those described by Virchow,* Dr. Bastian,† and others, were found situated on their walls.

An examination of the few cases here recorded, though not furnishing sufficient material for proving any pathological view, affords, I think, ample ground for declining to accept the one put forth by Professor Empis,‡ and others, that 'La

* Op. cit. Band ii. S. 632. † 'Path. Trans.' vol. xviii. p. 21.
‡ 'De la Granulie,' par G. S. Empis. Paris, 1865.

Granulie' is a disease by itself, frequently coexisting with tuberculosis, but distinct and capable of separation therefrom, not only by pathological changes recognisable after death, but even by clinical symptoms present during life.

The cases here recorded show also, what is often lost sight of, that tubercular meningitis is by no means confined to childhood or early youth; four out of the fourteen occurring in persons over 25, three between 20 and 25, and five in children under 10. One only of the whole number (XXIX.) could be said to have had phthisis; in none of the others had the lung tubercle, when present, commenced to soften. Le Diberder * also remarks that the most advanced condition of cerebral tubercle in his cases did not correspond with the most advanced lung mischief.

In three only (XXII., XXVIII., and XXIX.) was tubercle met with in the lungs in other than a miliary condition; in two of these (XXII., XXIX.) it was associated with granulations on some of the serous membranes.

The five cases XXI.–XXV. form a group by themselves, presenting the common characters of tubercular meningitis in children, and require no comment, except that in two cases the illness was attributed to violence; and this is, as far as my experience goes, exceedingly common among the poor. It is quite conceivable that a blow or fall of sufficient violence to cause slight concussion, or even temporary derangement of the circulation, might be the actual exciting cause of the brain mischief, but in the absence of any proof of injury to the head, such as an external bruise or scar (vide Case XXV.), one must hesitate before admitting the alleged fall or blow as the real exciting cause; and it must be remembered that the ignorant among us always like to refer their illnesses to some definite cause which is within their own comprehension. I do not know whether among the educated classes the history of blows and falls in connection with tubercular meningitis is equally common; but I have noticed among mine own acquaintances who have curvature of the spine that the deformity with them, as with the poor, is always ascribed to accident—a careless nurse who 'let me drop,' or 'a fall out of bed when a baby.' Parents and patients alike being most unwilling to admit the possibility of such deformity depending on 'anything constitutional.'

Case XXVI. differs only from those preceding it in the

* Le Diberder, 'Essai sur l'Affection tuberculeuse aiguë de la Pie-mère chez les Adultes.' Paris, 1837.

greater age of the sufferer, and in the obscureness of the symptoms during life—vomiting and slight inequality of the pupils being the only cerebral symptoms present.

Case XXVII. was the only one in which masses of tubercle were met with in the brain itself, and were associated with firm, hard, yellow tubercular exudation on the surface of the cerebellum. At the same time granulations were found on the membranes at the base of the brain, and the fornix and septum lucidum presented the softened condition so common in ordinary tubercular meningitis. The pleuræ and peritonæum were studded with granulations, and miliary tubercle only was present in the lungs. In this case, notwithstanding the amount and gravity of the brain lesions, the cerebral symptoms were slight, neither loss of consciousness nor delirium occurring.

In Case XXVIII., again, abundant granulations were met with on the peritonæum, whilst the lungs were stuffed with masses of tubercle the size of hazel-nuts or larger, and firm, hard, yellow tubercular exudation was found beneath the membranes of the brain. Case XXIX. was one of chronic rather than acute tuberculosis.

The five remaining cases are all interesting as examples of acute tuberculosis in adults. In all but one both granulations and lymph were found on the membranes of the brain; in one (XXXIII.) the naked-eye appearances were very slight, but under the microscope the pia mater was seen to be extensively and generally affected.

Among the adults, with the exception of cheesy tubercle in one instance, and cretaceous masses in another, in the bronchial glands, there was no evidence of any tubercular changes of old standing.

The rarity of tubercular affections of the pericardium makes its presence in Case XXXI. noteworthy. Nearly all pathologists have noticed the rarity of tubercle on the pericardium, Cruveilhier* being of opinion that its rarity was due to the small amount of subserous tissue; and the rarity of its occurrence with tubercle in the lungs is mentioned by Louis. One of the three cases of tubercular pericarditis recorded by Dr. Bristowe,† was somewhat similar to the present case, granulations being found on the surface of the pericardium. The pathological changes noted by Dr. Burrows,‡ in two of his cases of tubercular pericarditis do not seem to me quite conclusive evidence as to the nature of the disease, and in his

* 'Anat. path. gén.' p. 683. † 'Path. Trans.' vol. xii. Case 3, p. 63.
‡ 'Royal Med. and Chir. Trans.' vol. xxx. p. 77.

third case the diagnosis was not confirmed by a post-mortem investigation. Professor Empis * has never met with granulations on the pericardium, and the changes associated by Rokitansky with tubercle on the pericardium, differ widely from those here noted. In Case XX. the granulations and adhesions on the pericardium were identical with those on the right pleura, both to the naked eye and when submitted to the microscope.

The histories and clinical observations recorded in the foregoing cases are, I regret to say, very imperfect. Still they afford an illustration of the ambiguous and contradictory nature of symptoms; many, usually regarded by authorities as distinctive of different forms of cerebral disease, were absent, as, for example, retraction of the abdomen, which was oftener absent than present in the cases of tubercular meningitis; delirium in Cases XIII. and XVII. where acute arachnitis existed, and though present in XVI., it was not of that furious and maniacal character described by some as pathognomonic of meningeal disease. Vomiting (omitting as irrelevant Case XVIII.) was an almost constant symptom, being noted as present in fourteen instances; in four of the remaining cases it was either not present at all, or so slight as not to have attracted notice (XVII., XX., XXIX., XXXII.); whilst the histories of four (XIX., XXII., XXIII., XXIV.) are either wholly wanting or imperfect.

ARTICLE X.—*Notes on Diseases of the Joints, based chiefly on cases that have occurred in the Hospital during the present year.* By F. HOWARD MARSH.

THE conviction is daily gaining ground that joints are capable of, and indeed naturally prone to, a much greater amount and perfection of repair than they have generally been supposed to admit of. For, treated with rest long continued and as absolute as it can be made, first by a splint to prevent movement, and secondly by continuous extension by means of a weight, to keep the articular surfaces from being pressed together during spasm of the surrounding muscles, the majority of cases of injured joints that are admitted into the Hospital, and many cases also of even advanced disease, at least in young persons (under the age of 25), are now well and readily cured. And clinical experience of the reparative power of joints is

* Op. cit. p. 71.

confirmed by what is found in dissections. I have carefully examined all the joints that, in the past year, have been removed at St. Bartholomew's either by amputation or excision, and it has appeared remarkable, even in those worst instances (Cases VI., VIII., IX.), in which it has been imperative to amputate the limb, in order to save the life of the patient, how limited has been the disease, and how much reparative action has existed side by side with the process of destruction. The following cases, the greater number of which have been under observation in the course of the year, may illustrate these remarks. The first five are clinical; the remainder give the results of dissection.

Case I.

In this case the knee-joint was laid wide open during the removal of a large tumour that grew in the rectus tendon, just above the patella. It is related at some length, for it offers many points of considerable surgical interest. A spare and delicate-looking woman, aged 31, came to Mr. Paget at the Hospital, in September 1867, complaining of a bad knee. There was found, growing either in, or upon the patella, a tumour somewhat larger than a hen's egg. Its surface presented many low indistinct lobes; in some parts it felt nearly as hard as bone, but in others, chiefly over the lobes mentioned, it was soft and elastic. No movement could be detected between the tumour and the patella, but the two together, however related to each other, moved freely upon the condyles of the femur. The skin over the mass was healthy and non-adherent; no disease could be found elsewhere in the limb; the femoral glands were not enlarged; the muscles in front of the thigh were wasted, but not excessively so. The patient said that seven years before her knee had grown weak and painful after exercise, and she noticed a slight enlargement, as she thought, of her knee-cap. In the following five years the joint became gradually more disabled, and was often swollen and painful: but throughout this time the tumour itself altered very little. During the last two years, and especially within the last six months, it had enlarged quickly. She was now unable to walk upon the limb. Several of the surgical staff examined the case with Mr. Paget, and the general belief was, that the tumour had grown within the patella, expanding it, and here and there absorbing, and then projecting through, its walls. It was thought to be either a myeloid or an enchondroma: its

features were most unlike those of a medullary disease. But whatever its nature, there seemed no doubt that it should be removed. When the patient had rested a few days in the Hospital, Mr. Paget exposed and cut into the mass, and found it to have the naked-eye appearance of a fibrous tumour growing amongst and expanding the fibres of the tendon of the rectus. The patella was buried beneath its lower part; and above, and at the side of this bone it was separated from the joint only by a very thin layer of synovial membrane that was closely fastened upon its surface. As a tumour thus placed could not be removed without opening the knee-joint, Mr. Paget cut it and the patella clean out, and with them the subjacent synovial membrane, thus laying the articulation widely open in front. Then the skin was replaced and sutured, and the limb fixed upon a back splint, and 'swung' beneath a cradle. Of course, after such an operation the patient was for many days in great risk of grave complications; but these she escaped. The wound healed without any great inflammation of the joint, and without any serious constitutional disturbance. The treatment consisted in carefully maintained rest; irrigation of the wound by a constantly-flowing small stream of luke-warm water, the application of carbolic acid lotion, and in a carefully planned diet. At length, anxious to go home to her family, the patient left the Hospital with the wound healed and apparently sound, and with the limb in a perfectly good position. In a fortnight, however, she had pain in, and then enlargement of, the knee, and a new growth of the disease developed itself in the tissue of the scar. She returned to the Hospital, and amputation through the thigh was performed, but she died of exhaustion. On examining the original tumour under a microscope it was found composed of a firm stroma of fibrous tissue—apparently the remains of the expanded tendon, in the meshes of which were crowds of cells of various shapes and sizes, and for the most part presenting a very distinct nucleus, and which were from these characters believed to be of a malignant nature. On dissecting the knee after its removal, a large mass of encephaloid cancer was found in front of the lower end of the femur, and also infiltrating the bone, and hollowing out the condyles into large irregular spaces. At the post-mortem examination secondary deposits of cancer were found in one rib, and in the body of one of the dorsal vertebræ, but not elsewhere.

Case II.

For this case I am indebted to the kindness of Mr. Paget and Mr. Blaker of Brighton. The former saw the patient after his recovery, the latter attended him, and furnished me with the following notes. A gentleman, aged about 35, in the best of health, was about to plunge into a large swimming bath from the top of the dressing-room, which was covered with thick glass; the glass broke, and he fell through. The knee-joint was laid open by a clean cut across its front aspect, dividing all the soft parts and the ligamentum patellæ, and bringing into view the crucial ligaments, semilunar cartilages, and the condyles of the femur. None of these parts seemed injured; there was no foreign body in the wound, and little or no bleeding. The wound was accurately brought together by silver sutures deeply applied, and the limb was placed on a pillow and supported by sand-bags; iced water was constantly applied. The whole wound healed by first intention without a single bad symptom; and he returned to his home about six weeks after the accident. He is now able to walk, hunt, or take any other exercise with little or no impairment of the limb.

Case III.

R. T., æt. 55, a carpenter, very intemperate, and often very poor, while using a small hatchet accidentally struck his left knee at the inner border of the patella, making a wound two and a half inches long, which bled freely. When he was brought to the Hospital an hour afterwards, blood distended the cavity of the joint, and still flowed from the cut. As if to prove how grave was the injury to the articulation, there was found in the clot that lay in the wound a piece of articular cartilage about as big as the transverse section of a garden bean. It was supposed that amputation was imperative, but Mr. Savory, who saw the patient shortly after his admission, taking into account the great danger to life of primary amputation through the thigh, and the unhealthy appearance of this man, held that the wiser course would be not to amputate, but to take the chance, small though this seemed, of a successful treatment of the wound. Therefore, as much blood as could be easily displaced from the synovial cavity was removed; the limb was placed on a back splint, and cold dressings were applied. It is not necessary to give all the details of the case; an outline of it will be enough. The patient seemed for a time considerably re-

duced by the loss of blood, his skin was relaxed and wet, and his pulse soft. On the fifth day he had symptoms of delirium tremens; but he took eight ounces of wine daily, and the attack passed by. For many days his condition was made critical by threatenings of active suppuration in the joint, of delirium tremens, and of exhaustion; but these perils he escaped. Then he encountered through many weeks the risks that seemed from time to time about to be realised, of chronic disease of the joint; but at length he recovered and left the Hospital with a stiffened knee, but with no symptoms of remaining disease. The treatment consisted in: (1) rest, secured for the joint by a back splint; (2) occasional local depletion by leeches; (3) carefully chosen food and medicine, including wine and cinchona bark; (4) towards the end of the case strapping of the knee with the cerat. hydrarg. comp. (Scott's dressing).

Case IV.

A young Scotchman was under the care of Mr. Savory in the Hospital, with a loose cartilage in the left knee. The body, which could be found only with difficulty, although it caused very severe symptoms, was one day discovered at the inner margin of the patella, and at once removed by direct incision. It was as big as, and in shape like, an almond. The limb was confined to a back splint, and the patient to his bed. The wound healed rapidly, and with no appearance of undue inflammation, and in three weeks the man was discharged quite well.

Case V.

In 1864 I saw Mr. Coote extract by direct incision, from the knee-joint of a boy æt. 15, a loose cartilage that was flattened and disc-shaped, nearly circular and as big as a halfpenny. The wound healed rapidly, and without any inflammatory action.

Here, then, are five cases—and several others might be related—in which a large joint (the knee) was laid open, and in which a good recovery was secured. In two the patient was over 30, in a third 55 years old.

I remember a case that occurred under Mr. Skey in 1862, in which the patient, having jumped out of the window of a house that was on fire, sustained compound fracture of one leg and of both ankle-joints, along with extensive injuries of the surrounding soft parts. He recovered with the joints stiff; but so well

repaired that he has ever since been able to walk about and follow active business as a draper. A man was, this year, under the care of Mr. Paget, in whom the ankle-joint had been torn open and the foot dislocated outwards. The limb was placed on a splint, and extension made by means of strapping placed on the dorsum and sides of the foot. He recovered and left the Hospital with a joint that was stiff, but free from deformity, and which there was every reason to suppose he could soon walk upon.

A boy, æt. 12, was brought into one of Mr. Savory's wards in April last with a wound into his knee-joint half an inch long, from which synovia was slowly draining. His limb was placed upon a back splint and swung, and the knee covered with cold dressing. Acute inflammation of, and suppuration in, the joint took place, and for some days the boy was in a state of severe surgical fever. A free incision was made into the joint, with immediate relief of his symptoms. Pus was discharged for about a fortnight, the mischief subsided, and he was discharged ten weeks after the accident with a limb stiffened but otherwise apparently sound.

Case VI.

A boy, æt. 9, broke his fibula a little above the outer malleolus, and thinking that he had only sprained his ankle continued to 'limp' about as well as he could. At the end of a week acute inflammation occurred around the fracture, the lower part of the leg became swollen and painful, and its skin red, tense, and shining, and he was admitted into Darker's Ward under the care of Mr. Paget. Free openings were made for the discharge of matter, and through one of these it was learnt that a considerable portion of the fibula above the fracture was necrosed. As the boy was suffering severely from surgical fever, and was quickly becoming hectic, Mr. Paget removed the dead bone, in the hope that, rid of this source of irritation, the wound might become more healthy. This hope, however, was not realised, and three or four days later the ankle-joint, which was evidently now distended with fluid, was laid open by a free incision, and a large quantity of pus escaped. For a short time the lad seemed better, but his improvement did not last, and in order to save his life amputation (through the knee) was performed five weeks after the injury, and one month after the ankle-joint was observed to be involved. He recovered.

Dissection of the ankle-joint.—The synovial membrane lay around the margins of the articular surfaces, bright red and succulent, and for the most part, but not without exception, it had deeply eroded the cartilages wherever it came in contact with them. Almost every trace of cartilage was gone from the surface both of the tibia and the astragalus—small portions only remained here and there at the extreme articular margins of the bones; and here it was undergoing removal (1) at its free surface by the vascular fringe of synovial membrane that overlaid it; and (2) by the vessels of the bone beneath, for it was deeply undermined, and its deeper layers were already absorbed. With the microscope, the cartilage that remained was seen to be little changed; its matrix was too coarsely granular, its cells too numerous, and many contained fat. The bones were very acutely inflamed, and of a bright red colour on the surfaces from which the cartilage had been removed. In the centre and over the whole extent of the articular surface, omitting only the extreme margins, the astragalus and tibia were already so firmly fixed to each other that when the joint was opened a thin scale of bone had broken off the astragalus and remained fastened to the tibia. On trying to separate this scale I found that, towards the margin of the articular surface, it was connected with the tibia by soft gelatinous lymph, while towards its centre the union was by a firm, hard substance like bone. The surfaces of both the tibia and the astragalus were intensely red, cancellous, and somewhat softened. The redness and softening, however, were quite superficial, extending, as a section showed, not more than a line in depth, the remainder of the bone being pale, firm, and apparently healthy. In this case, within one month, there had occurred acute inflammation of the ankle-joint, with absorption of its cartilages, and then nearly complete bony anchylosis.

Case VII.

A. S., a puny, small-limbed seamstress, 21 years old, was received into Sitwell Ward, under the care of Mr. Callender, with chronic disease of the ankle and tarsus. The arch of the foot was completely lost; two sinuses led to bare bone in the tarsus, and there was considerable swelling on both the inner and outer aspects of the ankle-joint. The patient stated that her disease, which had existed for seven years, had never been very severe; but, though she had tried many things, and had rested her limb on several occasions for periods of various

duration, both at home and in hospitals, she had still remained subject to pain whenever she used the foot, and had long been prevented from gaining, as she might otherwise have done, a good livelihood. She begged that amputation might be performed. When she had been watched for some weeks, and there appeared but very tardy improvement, Mr. Callender, considering her poverty and the long time that must pass before she could regain the use of the foot, amputated through the lower third of the leg. The patient rapidly recovered, and soon left the Hospital. On dissecting the limb, I found disease of many of the tarsal joints, but this was, as the history of the case had indicated, nowhere either extensive or active. The astragalus was firmly anchylosed by bone to the tibia, and so were the tibia and fibula to each other.

Case VIII.

In May 1866, a man, æt. 28, who had previously been healthy, had acute synovitis of the right knee. He kept in bed, and was well at the end of a month; but in August of the same year he had a relapse, and from that time constantly suffered from a slowly-advancing disease of the joint. In April 1867 he was in the Hospital, under the care of Mr. Willett, with chronic synovitis, the joint then containing some fluid, and the synovial membrane being thickened. With rest he was soon so much better that he was discharged. He was again admitted in November (1867) and was then suffering, in addition to the chronic synovitis which he still had, with suppurations around the joint. He had, however, little pain and no other urgent symptoms. Subsequently he had an attack of very acute inflammation of the joint, and with it such severe constitutional disturbance that amputation through the thigh became necessary. He recovered.

Dissection of limb.—There was no deformity of the joint; its interior contained pus. The synovial membrane was intensely vascular, and very succulent and glistening. Under the microscope it was found occupied with exudation cells which were rapidly undergoing development into fibrous tissue; very few contained fat granules. The semilunar fibro-cartilages were soft, pulpy, and ragged. The articular cartilages had almost completely disappeared. The bones were exposed, vascular, and softened—for the most part they had suffered no further change; but here and there were small districts in which a thin layer was scaling off, and when these scales were

turned up there was found beneath a very dark, vascular, shallow (ulcerated) spot; but there was no softening or structural change except in the superficial layer of the bone. Even the surface looked in places scarcely at all different from healthy bone. In many places the surface of the bones was covered with lymph which was rapidly developing into fibrous tissue, some of which was already being converted into bone.

Case IX.

R. D., æt. 30, was admitted in November last, under Mr. Coote's care, with chronic disease of the knee, the result of a blow received some months previously. His constitutional symptoms became so formidable that amputation through the thigh was performed. For a few days he rallied, and it was hoped he would recover, but he died of exhaustion.

Dissection.—The synovial membrane was injected and swollen. The cartilages had almost completely disappeared. The bones were covered with a layer of lymph, which, in many places, was firmly adherent, and was rapidly being developed into well-formed fibrous tissue, producing fibrous anchylosis. When the fibrous material was torn away the surface of the bone was seen thickly set with delicate perpendicular pillars of new bone, looking like the pile of velvet, and which, jutting up into the fibrous tissue, were rapidly converting the fibrous into bony anchylosis. The bones were very little altered except upon the surface: here they were bare and cancellous; immediately below the surface they presented on section traces only of increased vascularity, but they were not softened.

These cases, I think, are sufficient to show that there is no essential deficiency of reparative power in the structures that form joints, and to hold out every encouragement, that when patients become more and more aware of the great importance of at once seeking surgical aid in cases even of slight injury or disease of the joints, and when such cases are, as soon as they are seen, placed under the conditions of perfect rest, practice in this department may become much more successful than it has hitherto been.

On the value of continuous extension by means of a weight, in the treatment of joint disease.

In a paper in the second volume of the 'Reports' I invited special attention to the point which has often been insisted on, especially by Mr. Hilton,* that the evil progress of many cases of joint disease is due mainly to pressure of the articular surfaces against each other, which is produced (1) by the spasmodic contraction of the surrounding muscles, and (2) by movement of the limb; and several cases were related in which all the symptoms had quickly ceased when the joint surfaces were kept apart by continuous extension secured by means of a weight suspended from the extremity of the limb. Subsequent experience has strongly confirmed the confidence with which I ventured to advocate this method of treatment. About eighteen months ago, some ladies who had previously for three years given their services as lady-superintendents of the wards of the Hospital for Sick Children opened the 'House of Relief' in Queen Square, Bloomsbury, for the reception of children, between the ages of two and ten years, who, having hip-joint disease and having failed to obtain admission into the Children's Hospital, might be received, and allowed to remain through any period necessary for their cure. Having visited these cases, which now number thirty, almost daily, I have had a good opportunity of watching their progress.

As a very general rule, in cases of acute disease, the weight speedily relieves pain and brings down the limb into a straight position, and it is a common observation that children, who while they wear a weight are absolutely free from pain, will, if the weight is removed, relapse within a few hours into their old suffering from spasmodic jerkings of the limb. But the following cases which, except the last, have occurred at St. Bartholomew's in the present year may best illustrate this method of treatment.

Case X.

A boy, æt. 14, while skating last winter fell heavily on his right hip. Symptoms of acute inflammation of the joint were rapidly developed with great severity; the limb was drawn up towards the abdomen; pain was very acute; there were frequent twitchings and jumpings of the limb, so that the boy could not sleep; he lost flesh quickly. Three weeks

* 'Lectures on Rest and Pain,' 1863.

after the accident, his condition becoming each day more distressing, he was brought, placed on a bed in the bottom of an omnibus, to the Hospital, to be under the care of Mr. Paget. When it was proposed to move him, his cries were such that chloroform was administered as he lay, and then he was carried up into Darker Ward. The treatment ordered by Mr. Paget was simply that of extension by means of a 3 lb. weight. The limb readily subsided into full extension, and his pain, although for the first few days but little relieved, gradually ceased, and within a fortnight had completely left him. During the treatment, 4 lbs. and then 6 lbs. were substituted for the original 3 lbs. He remained in bed for about eight weeks in all, and at length left the Hospital having long been free from pain. The limb remained stiff, and he was ordered for the present not to attempt to walk upon it.

Case XI.

A. B., an Irish labourer, æt. 56, was admitted into the Hospital, under the care of Mr. Paget, with acute inflammation of the knee following fracture of the patella. The joint was hot and extremely tender; he was constantly disturbed by muscular twitchings when he tried to sleep; any movement of the bed gave acute pain. He was losing flesh, had no appetite, had sweating, and looked pale and worn. His limb was placed upon a pillow, and covered with a large linseed poultice. He gained little relief, and his state grew daily more critical. Mr. Paget now directed that the limb should be placed upon a back splint, with a weight of 5 lbs. suspended from the foot. The man's relief was immediate. In the first night he was much less troubled with startings, and he had some good sleep. It is sufficient to state that thenceforth his pain steadily and rapidly subsided, and with it the tenderness. In a fortnight he was able to sleep well, and his pain was very trifling and only occasional. He remained in the Hospital for some months with a stiff crippled knee, but its condition slowly improved, and he at length was discharged, able to walk pretty well.

Case XII.

A. R., æt. 35, was attacked with inflammation of the right knee-joint a fortnight after her confinement in 1867. For nine weeks she remained at home using poultices, liniments, &c.,

but as the joint grew more and more painful she came into the Hospital under the care of Mr. Savory. On her admission into President Ward, there were the symptoms of advanced disease. The leg was semiflexed upon the thigh; the tibia was considerably displaced outwards and backwards towards the ham; the joint was much enlarged and extremely tender. Pain was not severe when the limb was at rest, but any disturbance of it, either by movement or even by the jarring of the bed caused acute suffering. The limb frequently started at night, and she got but little sleep. She was losing flesh quickly, and had copious night perspirations. For the first few days after her admission, the limb was placed upon the ordinary back splint and 'swung,' and the joint covered with a poultice, but her symptoms continued unabated. Continuous extension was then employed. In order that the weight (5 lbs.) suspended from the foot might act efficiently, the limb was placed upon a back splint, so contrived that the leg-piece could separate, in the direction of the long axis of the limb, from the thigh-piece, so that the weight tended to separate the articular surfaces of the femur and tibia. During the first night of this new treatment the patient was almost absolutely free from pain, and she said next morning that 'it was the first night's rest she had had for nine weeks.' From that time forward she had no severe pain, and the spasmodic startings soon completely ceased. When the weight had been used for a month, and when, from the absence of symptoms, it was thought she would no longer want it, it was removed. For a night or two all went on well, but then the pain and startings returned. The weight was reapplied and pain at once ceased. Treatment was continued for two months more, and then it was found that she could dispense with it. She left the Hospital at the end of fourteen weeks with a stiff but painless limb, and in perfectly good general health.

Case XIII.

Emma L., a bookfolder, æt. 22, was attacked with inflammation of the knee in the course of rheumatic fever, in the middle of last January. The joint became considerably swollen, and tender, and so painful that she could sleep very little. Her rest was broken especially by jumpings and startings of the limb. For six weeks she remained at home and in bed, and then, as each day found her worse, she was taken into the Hospital to be under the care of Mr. Savory.

The joint at this period was distended with fluid, the skin over it was tender and red, but there was no displacement of the bones. The limb was placed upon a back splint, and 'swung' beneath a cradle. She was soon more comfortable and able to sleep better at night; yet at the end of a fortnight pain from muscular spasm was occasionally very severe. A weight of 5 lbs. was now suspended from her foot. The benefit from this measure was immediate and most marked. She lost all pain, and her general health quickly improved. When nearly a month had elapsed without any recurrence of pain the weight was removed. Within a few hours muscular spasm returned, and she could get no sleep. The weight again speedily and completely relieved her. Subsequently on two or three occasions attempts to discontinue the use of the weight led to a return of spasm, but at length she could dispense with it. She left the Hospital at last with the joint apparently sound.

Case XIV.

In April of last year I had an opportunity, through the kindness of Mr. Paget and Mr. Nowell Stowers, of Kennington, of seeing a little girl, æt. 7, who had been suffering for a twelvemonth with disease of the right hip-joint. I found her lying on her left side, with the thighs drawn up towards the abdomen. She was, and had been for some weeks, in great pain, especially in the night, when startings of the limb spoiled her rest; she was thin and pale, and had no appetite; she screamed and evinced great terror when she was moved, and constantly kept her hand upon the thigh as if to control spasm of the muscles. A weight of 3 lbs. was suspended from her foot. In a few days she lost her pain, the limb subsided into full extension, and she could lie constantly upon her back. She continued to improve, and gained flesh and colour; pain remained either altogether absent, or only slight. The weight was worn for six months, and then a leather shield-splint was moulded upon the joint, and she was sent to the seaside, her parents being especially cautioned that she must still be kept lying down, and that she must not be allowed to walk, or to be in any way roughly or carelessly moved. On her return at the end of a month she was still kept chiefly on her back for four months longer, and then, after having worn the weight for six months, and subsequently lain in bed for five months, she was allowed by degrees to walk about the room. This is her present state: The limb as she lies in bed is in a perfectly

straight line with the trunk; there is no twisting of the pelvis and no abscess; the limb is shortened to the extent of rather more than half an inch; there is considerable movement at the joint. By means of Nelaton's line it is found that the trochanter major lies about half an inch above its natural level, so that it may be concluded that the shortening is due to partial absorption of the head of the bone and of the upper border of the acetabulum, and consequent drawing up of the femur.

On the treatment of hip disease.

The principles on which any disease is treated must depend on the view that is taken of its nature. Is, then, hip disease as it is so commonly seen in childhood a merely local disease, or is it a result of the strumous constitution of the subjects of it? Unfortunately 'struma' is at present a very indefinite term, and there is so much uncertainty as to its meaning that those who use it must first say what they wish to convey by it. I will do this as briefly as possible. Struma is different from tuberculosis; for the latter includes all those cases in which there is a deposit of tubercles in any part; and no case can be called one of tuberculosis, except in the belief that tubercles are or will be deposited. Struma and scrofula are in every way convertible terms. What is termed 'struma,' or scrofula, can be defined at present only by an enumeration of its reputed symptoms.

It is a defective condition of the general health, with which is associated a tendency to lesions of an inflammatory character, affecting especially the skin, mucous membranes, glands, and the cancellous texture of bones. This tendency to inflammation depends, according to Virchow, on an abnormal irritability or 'vulnerability' which leads to its development after trifling injuries, such as in healthy persons would have no consequence. In many instances inflammation originates in causes so slight that they escape observation, and then it is called 'spontaneous.' Thus a cold wind will give strumous patients an eczema or an ophthalmia; or a slight blow or sprain will be the starting-point of caries of some of their bones. Thus regarding struma, I cannot but believe that, speaking generally, hip disease must be regarded as strumous.

Cases are often seen in which the tendency to low forms of inflammation is shown by the occurrence, in the same patient, of disease in two or more of the large joints, as in both hips, or in a hip and an elbow, or a knee; and also by the develop-

ment of disease spontaneously, as in those cases in which, while a child is confined to bed with an affection of one hip, disease has sprung up in the opposite joint. Many cases, too, are brought to the Hospital, in which the disease must be termed spontaneous, for there is no evidence whatever of such an injury to the joint as would lead to disease in a child of healthy constitution. It may perhaps be said that an injury may have been inflicted, but overlooked or denied; but the burden of proving this would rest with those who assert its occurrence, and, of course, the fact that the disease has followed an injury does not prove the absence of struma, for a hurt that would lead to disease in a healthy child would so much the more do so in one that is strumous. And how do these cases of hip disease differ from those of the ordinary low forms of inflammation of other joints—the knee, the ankle, and the elbow—or from 'caries' of the spine, or of the bones of the hand and foot? And if it be answered that neither are these strumous, but the result of neglected local injury, what remains to form the class of strumous affections?

It was formerly agreed by almost common consent, that hip disease was strumous; but more recently surgeons of the highest authority have expressed their belief that in very many instances it is not so, but that it has occurred in healthy children, purely as the result of a neglected injury.

May I venture without presumption to remark on these two opinions? Agreeing with the first, that hip disease is, in a large majority of cases, strumous, I think the conviction that it is so has frequently led to a false conclusion as to its proper treatment. For struma being a constitutional defect, it has been held that its manifestations must be treated by constitutional measures, while local measures are of comparatively little importance. These children have been thought too delicate for confinement to bed or even to the house; they have been sent to the sea-side, and have been ordered to take cod-liver oil and iron, and the local mischief has been left very much to itself, on the ground that as it was the result of a general fault, it could not be corrected by local means, and that it would be repaired when the general health was set right. Undoubtedly there are many cases of obstinate struma, which nothing will cure so well as sea air, or, next to this, country air; but in a large share of cases struma, as it is seen in the children of the poor, is a much less obstinate cachexia than it is reputed to be, and one that may be successfully treated in a hospital. It is not in the least necessary to send patients

to the sea, or to be constantly striving to procure them fresh country air. They would be the better for these advantages, but they can certainly do without them. Among the thirty children mentioned at p. 188, although many of them are strumous, if any such thing as struma exists, there are none who suffer materially from confinement to bed; and I confidently assert that there is not one in five hundred of this class of children who may not be safely confined to bed, or who will not thrive there for any time during which the surrounding air is as good as ordinary hospital air, and while he is well fed, and while his local disease is doing fairly well. The want of success that formerly attended the treatment of hip disease was in great part due to the fact that, in order to secure fresh air, which was considered absolutely necessary, the child's joint was never at even moderate rest.

On the other hand, I venture to think that some surgeons now-a-days, while they are right as to the treatment that should be adopted, have taken a wrong view of the nature of these cases. Finding that the majority do well, when treated on the assumption that they are merely aggravated local injuries in otherwise healthy children, they have proceeded to deny the strumous nature of the affection, on the ground that, if struma were at the bottom of these cases, they could not be cured merely by local measures, but would require also constitutional measures. I would say that many of these cases are strumous (for the reasons given above), and that nevertheless local measures are sufficient along with good diet, and such fresh air as there is in hospitals, for their cure. That which is commonly called 'struma' is, in fact, a condition that is often created by deficiency of food and air, and by the neglect of the first principles of hygiene, and that may be cured by placing the subjects of it under mere ordinary sanitary conditions; and the proof of this proposition seems to lie in the fact that there is scarcely to be found a case of joint disease in childhood, except in very advanced stages, or of caries of the spine or of the tarsus and carpus, which will not continue to improve till it is cured, when once it is placed under proper local conditions, and is treated with local measures.

I hope it will not seem that this is merely a question of words. It appears to be of real importance. If it is held that, because these cases do well merely with local treatment therefore they are not strumous, it may be concluded that those other cases, in which the proof of struma is clear, will require constitutional rather than local treatment. The truth seems

to be that, strumous or not strumous, they all and equally require careful and prolonged local treatment, and that with this all tend to recovery, though doubtless they will do this at different rates of progress.

It is very advisable that a child with hip disease should have a separate bed. Even poor parents may provide this, for a wooden frame-work, which a carpenter would supply for a few shillings, will serve the purpose. A horse-hair or flock mattress should be used, and beneath it should be a board to make its surface firm and flat. It is better not to use a pillow, a child sleeps as well without as with one; and, as he is sure to lay his shoulders as well as his head upon it, it is mischievous in two ways—it makes him slide down towards the foot of the bed, and it prevents the limb from being brought into a straight line with the trunk. A cradle should be used, for the weight of the bed-clothes, if allowed to drag on the foot, will permanently evert the limb.

I may perhaps be pardoned for telling how a child with hip disease ought to be lifted. I refer to such apparently trifling details, only because I am convinced that the good of a fortnight's treatment is often undone by an unskilful movement of these patients. The nurse standing at the child's sound side stoops over him so that he can place his arm round her neck; she then puts one arm beneath his loins, and with the other, passed beneath the sound thigh, which thus rests on her forearm, she grasps the diseased thigh about at its middle and makes firm and carefully sustained extension, so that the joint surfaces are kept asunder. At the House of Relief it is found necessary, before a new nurse is permitted to move any of the children, to give her special instruction in this method of doing so. Children who make no complaint whatever when moved in this fashion, scream with pain when left in the hands of a novice.

I feel the greatest confidence in stating that in the very large majority of cases in which the disease is not far advanced—that is, before there is displacement, or long-standing muscular contraction, or abscess—continuous extension by means of an appropriate weight is the best treatment for hip disease that is at present known. I have watched its effect in a large number of instances, both at St. Bartholomew's and at the Hospital for Sick Children, and as a rule, to which there have been very few exceptions, muscular spasm and pain have very soon ceased, and, where the treatment has been persevered with, excellent results have been obtained. But sometimes collateral means must be adopted. Many children are restless, and some have night

terrors, during which they start up suddenly or toss themselves from side to side in bed. In such cases a long splint should be used. As a very general rule this should be placed not on the diseased, but on the sound side, for then the child will be more completely restrained, and there is no risk of injuring the diseased structures by pressure or bandaging, or of straining the joint by the dragging of the splint when the child is moved. A long splint, too, should be used when there is a difficulty in getting the patient to lie on his back, as it is necessary he should. In some instances, although the limb has been in good position and all other circumstances have seemed favourable, pain has continued to be severe. This has most frequently proved to be due to the formation of abscess in the joint, producing tension of the ligamentous structures, and it has in some cases continued in spite of all treatment, until the matter has either been let out by incision, or has made its way towards the surface. In other instances of deep-seated abscess the actual cautery has seemed by far the most valuable means of relief. It has often for a time completely and immediately removed very severe pain. Blisters have seemed much less useful than they are reputed to be. Frequently a succession of them has given no relief. The cautery should be applied lightly; a deep eschar is not required, and I have seen its free use in children in feeble health followed by pretty extensive sloughing of the skin. In a few cases pain has persisted without any obvious cause, and in spite of many combinations of treatment; but in one instance it was almost instantly removed by the application of a splint to the diseased limb.

As the disease advances it becomes complicated with deformity of the limb and abscess in or near the joint. The deformity that occurs is due in great part to the spasmodic action of the surrounding muscles. In some instances the thigh is simply flexed upon the pelvis, while in others the pelvis becomes twisted on the spine; or there is what is commonly termed dislocation of the hip. True dislocation, however, as Mr. Holmes* points out, is very rare in hip disease. The head of the femur and the upper margin of the acetabulum suffer absorption, and the trochanter major is drawn upwards towards the dorsum ilii by muscular action; and it is this condition of parts which is mistaken for true dislocation. It is not rare that the head and part of the neck of the bone are so completely absorbed that the upper part of the femur is little more than a

* 'Surgical Treatment of Children's Diseases,' p. 442.

direct continuation of the shaft, and then the limb is carried by it own weight, aided by that of the bed-clothes if these are allowed to drag upon the foot, into extreme eversion, so that the patella looks directly outwards. This deformity sometimes takes place suddenly, from the rupture of ligamentous fibres that previously restrained the limb. The pelvis may be twisted in two directions; it may lose its horizontal position, so that it becomes lower on one side than on the other, or rotated on the spine, so that the diseased hip projects backwards. In the early stage of disease the pelvis is lower on the diseased side, so that there is apparent lengthening of the limb; while, later, it is found raised on the diseased side, and rotated backwards on the spine, so that the hip is very prominent. These various deformities are due chiefly to muscular action; but the exact mechanism of some of them has not yet been fully ascertained.

Flexion of the thigh on the pelvis should be corrected as soon as possible. While it remains the limb is shortened, and it gives rise to a very serious deformity—lordosis as it is called—which consists of a complementary anterior curvature of the lower part of the spine in consequence of the effort of the patient to straighten the limb. That this curvature of the spine is produced in this manner is, I think, proved by the fact that if the patient, when lying on his back, tries to straighten the limb, lordosis is increased; while, on the other hand, it may be completely removed by raising the diseased limb to the requisite extent in the air. In some instances the real amount of flexion at the hip can only be ascertained by thus raising the limb till the spine is enabled to resume its natural outline. The limb may be safely brought down under chloroform. I have straightened at least fifty in this manner, and have seen many others so treated, and I do not remember that, when proper care has been used, any bad result has followed. In some instances it is necessary and best to bring down the limb gradually, that is by repeated operations, rather than to attempt to do so at once. It has often been found that a limb which yielded scarcely at all on the first trial has readily come down subsequently, probably because some inflammation has been set up in the adhesions, and has led to their partial softening. After the straightening a long splint should be placed on the sound side to keep the patient on his back, and a weight as heavy as he will bear (for a child five or six years old this may gradually be made six or eight pounds) should be suspended from the diseased limb. Straightening may, if necessary, be repeated at the end of ten days. The weight should be reduced

as soon as possible—that is, when it is found that there is no tendency to recontraction. American surgeons are in the habit of frequently cutting tendons in these cases, because they say that the muscles, from long contraction, undergo absolute shortening, and oppose extension of the limb. I have, however, in no single case found it necessary, or so far as I could judge advisable, to follow this practice, for I have not found any muscles particularly tense. Twisting of the pelvis is sometimes very insidious in its occurrence, and takes place when the patient is free from all other active symptoms of his disease, and it is sometimes very extreme. It is difficult to correct or to control it. The best method of treatment seems to be by a long splint adapted as closely as possible to the sound limb. In a few instances it has seemed useful to weight the foot of the raised side heavily; in one case extreme twisting was removed by placing the patient on her face for about three weeks. But I do not pretend either to understand the exact mechanism of this deformity in its different forms, or to point out its successful management. Before the patient is allowed to walk, on his recovery, if there is any shortening of the limb, he should be furnished with a high-heeled boot.

The formation of abscess is extremely common in the course of hip disease. Comparatively few patients escape it except those who are carefully treated at the first appearance of active symptoms. On referring to the notes of thirty cases I find that of these twenty-eight have had abscess. So far as I have seen this complication is less formidable than it is generally supposed; these formations are usually 'cold' and chronic, and although some give rise to severe pain by producing tension of ligamentous structures, and others attain considerable size, they do not appear to produce any severe constitutional disturbance, or to indicate extensive disease of bone. They are least mischievous when they are repeatedly opened with a small trochar, or by small incisions, so that their cavity may gradually contract.

It is scarcely safe to lay them open at once by a free incision and it cannot be necessary to do so. Many must ultimately, no doubt, be freely opened; but if they have been previously tapped with a trochar, they appear more tolerant of active treatment. It is better if possible to defer the opening of an abscess that has formed quickly with heat and tenderness of the surface, until it has subsided into a more quiet state. In a few instances matter has burrowed widely, and formed many sinuses, but in the majority these abscesses after being punctured

have discharged for a few weeks, or for three months, or longer, and then have soundly healed. Generally the puncture, even of large abscesses, has been followed by no constitutional or local symptom whatever. I have opened those that form in front of the joint, and which are in the neighbourhood of the trunk or branches of the femoral artery, by the method recommended by Mr. Hilton.* A small incision is made in the skin, and a director is carefully thrust through the soft parts, and into the sac of the abscess; as soon as matter is seen escaping by the groove of the director, a pair of fine dressing forceps is passed along its track, and the blades forcibly separated. Besides its safety, this method offers the advantage that the opening is not so likely to close as that which is made by a simple incision.

I have notes of two cases in which I have no doubt that collections of matter of considerable size (probably containing in the one three ounces, in the other an ounce and a half) have been absorbed. Of the abscesses that form in connection with hip disease, the most favourable are those that belong to the class which Mr. Paget has termed 'residual,' and which occur in tissues that have been damaged by a previous disease that is now soundly cured. A boy 12 years old suffered for eight years with hip disease, which, however, had never been acute. All his symptoms subsided, and he was apparently well for a year, when an abscess formed on the outer aspect of his limb. As he had no symptoms whatever of renewed disease, the abscess was opened, and it healed like an ordinary chronic abscess in the cellular tissue. He remains quite well.

A woman, aged about 30, was in Sitwell Ward three years ago, who had suffered from hip disease, and abscess on the outer aspect of her limb in childhood. The abscess discharged itself, and the disease was at length cured. She remained well for more than twenty years, and then, when her health was reduced by poverty and a recent confinement, a large abscess formed in the site of the former collection; but she had no symptom of the return of disease in the joint. The abscess was opened by free incision, and it very quickly healed. Her hip remained sound. A woman, aged 30, is now in Lucas Ward under Mr. Savory's care, who made a good recovery from hip disease in childhood. After being able to take very active exercise for twenty years, and having no symptom of recurring disease, she has now had a large abscess on the outer aspect of the thigh, which made its

* 'Lectures on Pain and Rest,' p. 120.

appearance when she had been reduced in health by nursing a relative. Other cases might be mentioned, but these will suffice to show the character of these formations. When they occur in connection with the hip they need excite no suspicion of a renewal of the disease; and they may be freely opened, and treated after the fashion of a simple chronic abscess.

Article XI. — *Surgical Cases.*

By Thomas S. H. Jackman.

Case I.—*Rupture of the popliteal artery—Mortification of the leg—Amputation—Recovery.*

Mr. WEBB, æt. 56, living at Lingin, a small village about five miles from my residence, a farmer of abstemious habits and always in the enjoyment of good health, was on January 24, 1862, superintending his men, who were removing some large stumps of old trees. The men had placed a chain round one while he was standing with his back to them, the horses employed made a sudden plunge forward, which tightened the chain with a jerk, and in so doing it caught him just under the knee and threw him down with much force. He felt great pain in the part struck and down the leg, and being unable to walk was carried home.

Upon my arrival, I found some swelling in the popliteal space, with discoloration of the skin caused by the bruise. The foot and leg were cold and somewhat paler than natural, but not in the least swollen. There was no pulsation to be felt in the anterior or posterior tibial arteries. He complained of much pain in the popliteal space and down the leg. There was also some constitutional disturbance. I ordered flannels wrung out in hot water to be applied to his foot and leg, and a cold spirit lotion to the popliteal space and knee. The swelling in the popliteal space became somewhat less, but the foot and leg remained cold and pale, with much pain, requiring the daily use of opiates.

There was no change, in fact, in the appearance of the leg or foot until February 3, when I noticed that the foot, more particularly the toes, had changed colour, and on the next day there was no doubt that gangrene had set in, and this spread rapidly up the leg.

On the 7th, I amputated the limb above the knee, Mr.

Covernton, of Knighton, kindly assisting me. The man made a very good recovery.

On examining the amputated leg, and carefully dissecting the popliteal artery, we found in the middle of its course a round tumour, about the size of a pigeon's egg or a little larger, into which the artery appeared to enter and from which it passed out a little below and at the side. On opening it, blood escaped. It had all the appearance of a false aneurism, and was no doubt formed from the fibrine of the blood between the broken ends of the artery. The walls of the sac varied a little in thickness from $\frac{1}{8}$th of an inch. The artery above was pervious down to the walls of this sac, and then it was effectually sealed; the lower end was closed for some distance and perhaps was impervious to the passage of blood from the time of the accident. The lining of the sac was quite smooth, and it was impossible to point out where the artery was connected with it.

CASE II.—*Strangulated inguinal hernia returned 'en masse'—Operation—Recovery.*

On February 22, 1857, I was summoned to Richard Pinches, agricultural labourer, living at Whitton, æt. 44, suffering from strangulated oblique inguinal hernia. He stated he had been ruptured three years, since which time he had always worn a truss, but that the day before, having broken it, he went this morning to work without one, and continued at his work, though in much pain, till mid-day, when he was obliged to return home, not being able to replace the rupture as he had always been accustomed to do. Vomiting came on and I was sent for.

I found him in bed with all the symptoms of strangulated hernia. In the right groin was an oblique inguinal rupture about the size of a hen's egg, pear-shaped and rather hard. I tried taxis and the hernia bounded out of my hand into the abdomen, not with the usual feeling of a reduced hernia; there was no 'gargouillement,' but with the sensation we might expect to be produced by an egg, or a ball surrounded by a wet bladder escaping from under the grasp of the hand. I feared what had taken place, that it had been reduced 'en masse,' but the man expressed himself as being much relieved, and the vomiting ceased, so I left him, directing him to remain in bed until he had another truss. About three hours after this, his wife came to me as I requested her, and reported that his bowels had been relieved, that he was easy, and that there had been no return of

the vomiting. I began to hope that my impression was wrong, but gave the wife strict orders to let me know, should any untoward symptoms occur.

The 23rd I heard nothing of him, but on the morning of the 24th between seven and eight o'clock A.M., I was sent for. He was suffering much from stercoraceous vomiting; this came on during the night. His countenance was anxious; pulse above 100, and small; tongue dry in the centre. He complained of pain in the abdomen, which was increased on pressure, particularly over the internal ring where there was a slight fullness. He had hiccough. The course of the inguinal canal was quite free from all swelling. He stated that the day before he had had much pain in the abdomen, which during the night increased, and vomiting then came on. He did not, as I requested, send for me, as he thought the pain would get better. I decided to operate, and sent to Ludlow for assistance. Mr. Valentine kindly came and rendered me the aid I required.

I dissected down to the external ring into the inguinal canal and passed my finger readily up to the internal ring, which was large, well-defined, and free; resting against it, within the abdomen, I felt what I doubted not was the hernia. I passed a pair of forceps up, and readily brought down the hernia. On opening the sac a little coloured serum escaped. The intestine was of a dark chocolate colour, but shining. I divided the stricture at the neck of the sac, which was tight, and after further examination returned it into the abdomen; the wound was treated in the usual way, a full dose of opium was given, and the man made a good recovery.

The cause of this hernia returning 'en masse' was doubtless the few, if any, adhesions that the sac had formed to the surrounding parts, and also the large internal abdominal ring. Certainly the force I used was but slight to what is usually required to reduce a strangulated hernia. I had, in fact, only just grasped the hernia when it slipped from my hand as described, and that this is generally, if not always, the cause of this rare accident, is shown by the fact that nearly all the recorded cases have been instances of reduction by the patients themselves.

CASE III.—*Fracture of the tibia and rupture of the anterior tibial artery—Amputation—Recovery.*

S. Morgan, æt. 60, living at Birtley, by occupation a wood-feller, a hale old man, sent for me on October 12, 1860, having met with the above accident from a tree striking him when falling.

When I arrived, about four hours after the accident had occurred, I found the limb very much swollen and very tender; there was extensive ecchymosis, and a peculiar livid discoloration over the foot and instep, which as it extended up the leg gradually faded away. There were many bullæ varying in size from a large walnut to a pea; the foot and instep were cold; there was no pulsation in the anterior tibial artery, nor could I feel any in the posterior; the swelling extended round and a little above the knee. The tibia was fractured obliquely at about its upper third. I put the limb in a McIntyre's leg splint and swung it, directing flannels wrung out in hot water to be constantly applied to the instep and foot, and a cold spirit lotion to the upper part of the leg.

The leg remained much swollen, and ultimately the whole of the soft parts around the fibula and between it and the tibia to within an inch of the external malleolus sloughed away. A larger abscess formed in the thigh, which, after much loss of time, I succeeded in persuading him to let me open. About three-quarters of a pint of very fœtid pus escaped.

I had constantly tried to prevail upon him to let me remove the limb, but he would not agree to it, saying he would rather die. He became hectic, and remained so for several weeks, during which time he took large quantities of brandy and other stimulants; but, on November 29, he having acquiesced, I, with the assistance of Mr. Thos. R. Adams, amputated the limb. His state at this time was such that had he not yielded to my persuasion he must have sunk in a few days. On this account it was out of the question to amputate above the knee. I placed him under the influence of chloroform, and dissected the soft parts from the inner side of the leg, and so got a fair flap to turn up over the end of the tibia and its outer tuberosity, and so come into apposition with the soft parts covering the knee. The fibula was perfectly dead and the articulation with the head of the tibia was destroyed, so that the whole of it was removed.

The man daily improved in health, and the wound healed with the exception of a fistulous opening behind, out of which

dead bone at times escaped up to a short period before his death, which occurred about eighteen months ago; but this state of the stump did not prevent his using a wooden leg. On his recovery his mind gave way, and it was necessary for him to be confined in a lunatic asylum, where he remained about three months, when he returned home well.

On examining the amputated leg and dissecting the arteries, we found the popliteal entire, but the anterior tibial was torn across within about a quarter of an inch of its origin.

Article XII.—*A Case of Œsophagotomy.*

By Alfred Willett.

THE operation of œsophagotomy is so seldom performed even in our large metropolitan hospitals, that I consider I am only executing a simple duty in placing the following instance of it on record.

I believe Mr. Cock's successful case of removal of a false tooth impacted in the pharynx, by incision into the gullet in the neck, is the latest case reported:* this occurred at Guy's Hospital in 1856. Many years previously Mr. Arnott performed the operation at the Middlesex Hospital on a child, on account of a fragment of bone having lodged for so long a time as to place life in danger. The patient at the time of the operation was suffering from pneumonia, which ultimately caused death.

These two cases are, so far as I can ascertain, the only instances of œsophagotomy performed in this country. Mr. Cock in narrating these two, supplements them with six others performed abroad, in five of which the operation was successful; so that, taken altogether, there have been six successful cases and two deaths. In all of them the operation was undertaken for the removal of foreign bodies.

The peculiarity of my own case is, that the operation was performed on a patient in the last stage of exhaustion from starvation depending on stricture of the gullet. Instances of the performance of gastrotomy as a means of alleviating the distress at this period of the disease have been brought before the profession, but I have been unable to discover any case in which œsophagotomy has seemed possible or has been attempted in a case of stricture.

* See 'Guy's Hospital Reports,' series iii. vol. iv. for 1858.

With this preface I proceed to relate the case of M. D., æt. 47, a female domestic servant, who was admitted into Lawrence Ward, St. Bartholomew's Hospital, under my care, June 18, 1867. A pale, spare woman, but not unhealthy in appearance, although evidently insufficiently nourished. She stated that for the past seven months she had experienced an increasing difficulty in swallowing, until at length she was wholly unable to swallow any portion of solid food, and could only get fluids down sip by sip. If she attempted to take a draught choking ensued, and the fluid would be immediately rejected. She was now so thin and weak as to render her unable to perform her duties any longer. Her family history gave no evidence of inherited disease, and her own health had been perfectly good until the commencement of the present symptoms; she had never previously suffered from sore throat, nor in her recollection had she ever swallowed any corrosive substance; her voice was slightly husky, and she always kept her neck very stiff. She had scarcely any cough but expectorated a frothy phlegm. There was no history of her having brought up blood or any kind of discharge. When asked to indicate the site of the obstruction, she grasped her neck behind the thyroid cartilage. In the process of deglutition, if the food once passed the obstruction, she felt quite certain it went onwards without further difficulty.

Although her neck was very thin and shrivelled, only negative results were obtained by an examination of it externally. No swelling or abnormal appearance could be detected, nor was there any evidence of the glands in the neck or elsewhere being affected, and no pain was caused by handling the parts.

On attempting to pass a probang, its progress was abruptly stopped at a point corresponding with the junction of the pharynx and œsophagus. After several failures I succeeded in passing a No. 8 bougie beyond the obstruction, and on looking at its point afterwards I found there was neither blood nor other discharge on it; lastly, I passed my finger down, and although I could not quite reach the obstruction, yet I distinctly recognised the pharynx becoming narrowed and puckered.

The treatment consisted in passing a bougie daily through the stricture, and in supplying her with as much fluid nourishment as she could take. Every day she had meat, which she liked to chew, as she obtained thereby great relief to her cravings of hunger.

When the stricture had been dilated so that a No. 14 bougie would pass through it an abscess formed in front of the cricoid,

apparently from the irritation of the instruments, which were directed to be omitted for a time; in fact, she was never able to bear their introduction again, as they produced so great an increase of irritation and suppuration. Progressive weakness, emaciation, and dysphagia continued without intermission for many weeks. The abscess did not close, the discharge being a watery purulent fluid, in which no trace of anything she swallowed was ever found, although occasionally bubbles of air were seen.

By the middle of October, four months after admission, she had become emaciated to the last degree, too weak to stand and only able with the greatest difficulty to swallow very small quantities of brandy and essence of beef; still she absolutely refused, as she had always previously done, to allow nutritive enemata to be given her.

After consultation with my colleagues, and with the patient's full consent, œsophagotomy was resolved on. The operation was performed on Saturday, October 19. Before commencing the incision a No. 5 bougie was passed down to the stricture and pushed onwards, the intention being that it should serve as a guide, on which to open the œsophagus when it had been brought in view. The point of the instrument, however, appeared beneath the integument on the left side of the neck, between the larynx and sterno-mastoid muscle, thus clearly showing the existence of a previous 'giving way' of the gullet, above the stricture. An oblique incision, nearly parallel to the inner border of the sterno-mastoid, from the lower border of thyroid cartilage to the sterno-clavicular articulation, having been made, and the platysma and fascia divided, the dissection was cautiously proceeded with; the depressor muscles of the larynx were either cut or drawn aside and the carotid vessels in their sheath were drawn outwards. After this some little difficulty was occasioned by numerous fistulous tracks and abscess-seats being met with, disguising the appearance of the structures, and deceiving one as to their nature; but by steadily keeping to the side of the cricoid cartilage and trachea, at length the muscular tissue of the œsophagus was recognised and cut into longitudinally; I introduced my little finger, found the œsophagus below quite patulous and healthy; above, however, my finger was at once arrested by a dense, unyielding stricture, thus showing I had opened the gullet immediately below the seat of stricture. The largest sized tracheotomy tube having been passed in and secured, a flexible tube was passed through it into the stomach without difficulty, thus proving the gullet

to be free from obstruction below. The bleeding was trivial and no vessel of importance was even seen, except the brachio-cephalic vein, which lay deeply across the lower part of the wound.

She rallied well from the operation, no sickness ensuing. For feeding her, a funnel and flexible tube were made to fit into the canula, and by this means essence of beef, milk, and brandy, in small quantities at frequent intervals, were administered. She was fed but sparingly at first. She slept well, was free from pain, and expressed herself as much relieved and quite comfortable. As there was but little febrile reaction, when forty-eight hours had elapsed, she was allowed nourishment without limit; indeed, for the first ten days after the operation the patient's progress was most satisfactory, gaining strength and losing that death-like appearance that had haunted her so long; but with increased power she became less tractable, insisted on trying to swallow when the tube was out, fancying she could do so more easily than before, and, although it was impossible such could really have been the case, she resisted the reintroduction of the tube and administration of food by it, consequently she did not get that regular and abundant supply which she required—in fact, for one whole period of twenty-four hours she successfully resisted every attempt to feed her by the artificial opening. A bed-sore forming at this crisis added another element of exhaustion. Somewhat rapidly she lost ground, showing no signs of rallying powers; and, gradually sinking from exhaustion, she died on November 5, the eighteenth day after the operation. A local cellular inflammation, apparently commencing in the bursa over the olecranon of the left arm, appeared two days before death.

The following report of the post-mortem examination, conducted thirty-six hours after death by Mr. Howard Marsh, is taken from the Hospital register.

Body.—Extremely emaciated and shrivelled; a recent bed-sore over the sacrum; the lower third of left upper arm and upper fourth of fore-arm swollen and of dusky red-brown tint, pitting on pressure.

Site of operation.—Wound extended from rather above the level of cricoid on left side to sterno-clavicular articulation; edges healthy, with no signs of infiltration or local irritation, the integument and muscles being easily separated from one another. The œsophagus and trachea having been removed and opened from below, a stricture of about an inch in length was seen, the upper border being opposite to the middle of the

cricoid cartilage. The stricture was formed by an infiltration of the whole circumference of the tube, but the chief interference with the patency of the canal was produced by a bulging backwards of the tissues between the œsophagus and the posterior aspect of the cricoid, these being especially the seat of infiltration. The canal was not only narrowed so that it would only admit of the passage of a No. 3 catheter, but was also tortuous. The inner surface of the œsophagus was ulcerated opposite the seat of stricture, and numerous fistulous tracks ran outwards into the soft parts around. Immediately below the stricture was a longitudinal opening into the left of the front aspect of the œsophagus, which readily admitted the finger; it traversed healthy parts, and the canal below it was perfectly natural. The canula that had been worn during life had caused no appreciable irritation. The mucous membrane over the arytenoid cartilages was in a state of (apparently chronic) œdema. On examining the part from the front, several old sinuses were found running from the stricture forwards among the muscles and opening into the wound. The innominata vein lay obliquely crossing the inferior extremity of the wound. Its coats were almost bare.

Heart extremely small, weighing only $4\frac{3}{4}$ ozs. Its coronary arteries were atheromatous (slightly), and both these and the veins on the surface of the heart were tortuous and dilated. This appearance was probably due to the extremely shrunken state of the organ, the vessels being rendered as it were too long. The heart's substance was extremely granular, and a number of fat globules lay about the field of the microscope.

Liver, spleen, and kidneys extremely atrophied only. No secondary deposit was found anywhere, and no enlargement or other change was found in the lymphatic gland system.

Microscopic examination of the stricture tissue.—Made up almost purely of cells of the squamous epithelial type, many were polygonal, as large as, and in fact just like, pavement cells, but many were oval or spherical, while others were tailed, long processes projecting from the oval as well as from the polygonal kinds. The cells were in almost every instance very distinctly nucleated.

Upon the above case I propose to make but few comments. The diagnosis of cancerous stricture of the gullet in an early stage, if not absolutely certain, was yet accepted as the reasonable and probable interpretation of the history of the patient, and the physical signs of her disease, and also that in the

limitation of the stricture to a small portion of the canal it followed its ordinary pathological conditions. With regard to the early treatment in the Hospital, I allow at once that the use of bougies was the cause of much mischief. Still, I believe I should not have been justified in omitting all attempts at dilatation. No external applications were used, except poultices when suppuration threatened, and of course I did all in my power by attention to the state of her general health to promote her comfort and sustain her strength. It is much to be regretted that the patient so persistently and obstinately refused to allow of the administration of nutrient enemata, for to this circumstance must be chiefly attributed her rapid emaciation and exhaustion. I am confident most surgeons will allow the propriety of operative interference, although some might have decided in favour of gastrotomy instead of œsophagotomy on the double grounds of the advisability of operating as far as possible from the seat of disease, as well as rendering it a matter of certainty of being below it. Still, feeling sure the stricture was of limited extent, and that I could open the tube below the stricture, I did not hesitate in advising œsophagotomy as the simpler and safer operation. But now the case is over, I confess I regret that the operation was not carried a step further and the strictured portion of the gullet divided. I do not thereby advise the needless interference with malignant structures, nor would I wish to press unduly a comparison between such a proceeding and the usual operation of perineal section for stricture of the urethra; still I feel strongly that in the performance of an operation of this character, a surgeon is bound to give his patient all possible benefit; and considering relief from starvation was the object here sought, and not the permanent cure of the patient, I am confident this poor woman would have experienced greater relief, would have been more hopeful of the future, and would, I firmly believe, have lived longer, if the power of swallowing had been restored to her. These cannot be considered trivial matters; they constitute the essence of a satisfactory result in such an operation, and to my mind far outweigh the possibility of hastening the progress of the disease. With an exhausted, well-nigh dying patient, such a contingency is surely of minor import. Looking back I lament the operation was not performed at an earlier date. I can only plead, as has so often to be done, when operations of expediency are under discussion, the extreme reluctance I had to ask the

consent of my patient to submit to a hazardous operation before the absolute necessity for it arrived.

Mr. Howard Marsh has so admirably reported the post-mortem examination, that no point seems to require clearing up; however, I cannot help calling attention to the perfect accord that appears between the features of the case as noted during life and as revealed at the post-mortem examination. I do not think there is anything, unless it be the extreme atrophy of the viscera, that could not have been foretold, and even this is quite in accord with our pathological experience.

Upon the general considerations of the operation of œsophagotomy, I do not know that this case casts any new light; our experience of it is still very limited. I found no difficulty in the operation, neither do I think there ever would be any, provided the site selected be not too high or too low. In thus limiting the range of the operation, I believe I am rather opposing the opinions expressed by Mr. Cock, who in his article in the 'Guy's Hospital Reports,' remarks on this point thus: 'However slender our experience may be, the feasibility of the operation is established by the fact, that it is quite possible to make a long deep incision along the side of the neck, and to continue the dissection down to the bodies of the vertebræ without compromising any vitally important organ or tissue.' Further on, he adds 'that a considerable longitudinal incision can be made into the gullet, whether pharynx or œsophagus, without present mischief or future detriment.' And again, 'I am inclined to believe that a foreign body might be extracted from any portion of the cervical gullet, including a range commencing above that point of the pharynx which may be inaccessible from the mouth, and terminating below at the upper opening of the chest. It might even be possible to extract a foreign body from the intrathoracic portion of the œsophagus provided it could be reached with the fingers, and thus brought under the influence of a pair of curved forceps.'

I take Mr. Cock's meaning to be, although it is not so stated very distinctly, that he believes any portion of the cervical gullet, within the limitations expressed, may be safely and surely cut into for the purpose of extracting an impacted foreign body. Considering how freely the gullet had to be divided in his operation, before the extraction of the body was effected, this view—viz. that the gullet may be cut into anywhere in the neck—seems to be the logical as well as consistent con-

clusion to be drawn from the article. I believe that the range within which the gullet can be opened is very much more limited. How high it may be cut into is never likely to require a practical solution; but how low down it may be possible to effect this step, I conceive to be a very important point to settle. To my mind it seems only necessary to remember that the termination below of the external incision is limited; that the œsophagus—always closely applied to the spine—commencing opposite the fifth cervical bone, almost immediately, in accordance with the spinal posterior curve, passes somewhat sharply backwards and becomes very deeply placed in the neck; that the great vessels all converge at the root of the neck, and on the left side the brachio-cephalic vein crosses over the trachea, often so high in the neck, as in my patient, as to form a bar to any further extension of the incision. In my own case, during the operation I wished to open the tube as low as possible, but from the causes mentioned above, chiefly from the œsophagus so quickly passing out of reach, I was forced to content myself with cutting into the gullet close to the level of the cricoid cartilage. Here the operation is easy, and I would strongly urge the selection of this site in any future operation. It may be said, that if the operation be undertaken for the extraction of a foreign body you must make your cut wherever that may happen to lie. To this argument I would answer, that, in the majority of the reported cases, it has been found impossible to do more than speculate upon the spot where the body has lodged; but surely of all places it will usually be there, where the large funnel-shaped pharynx contracts and merges into the comparatively narrow-tubed œsophagus—this is immediately behind the cricoid cartilage, the site I have alluded to. Even should the offender not lie immediately there, one difficulty, the gaining the gullet, has been overcome, and then with comparative ease the opening may be either dilated or further incised. The point I wish to press is the ease with which the gullet may be reached on a level with the cricoid cartilage, the difficulty that will be encountered if it be sought below it.

Article XIII.—*On the Molluscum Contagiosum of Bateman.* By Dyce Duckworth, M.D. Edin.

THE following cases of this disease having come under my observation in the course of the last two years, I have been induced to trace their history, and enquire how far the

element of contagion plays a part in the production of this cutaneous affection.

Some of the cases afforded me means of studying the pathological anatomy of this form of molluscum, and I have recorded the results of my examinations in this direction.

Case I.

A. A., æt. 9 years, was admitted into Elizabeth Ward with hydatid tumour of the liver, under the care of Dr. Edwards. She had a number of mollusca situated mostly about the mouth and clavicular regions. The hepatic tumour was treated by puncture to liberate the hydatid fluid, and the case did well. Nothing was done for the mollusca as some of them appeared to be drying up and disappearing.

About six months afterwards this patient came under my care at the Royal General Dispensary. Her mother thought the belly was again enlarging. Several of the mollusca remained, and from one of them I was able to squeeze the usual white pultaceous matter. Mr. Newstead bisected some of them and applied solid nitrate of silver to the surfaces. In a few days these had shrunk and were passing away. As the girl was badly nourished and tended, Dr. Church kindly readmitted her into the Hospital. The abdomen was carefully measured from time to time, and as it underwent no alteration she was discharged after a few weeks.

From the mother of this girl (an intelligent woman) I procured the following history—in the same house a baby about twelve months old had the same affection. This was the first case. Two or three brothers and sisters of this infant, of various ages up to nine years, subsequently suffered from it. A. A. (Case I.) was affected at the same time. Next, an infant sister, aged nine months, took it, and had 'the spots' for about ten months. Lastly, a brother, aged six years, became affected, and had three 'spots' which remained for about a year.

Case II.

A. C., æt. 3 years, a Jewish child, was brought to me at the Royal General Dispensary by the mother of A. A. (Case I.) to show a tumour of molluscum on the left infra-clavicular region. This occurred ten months after A. A. had been under my care. I detected another small tumour upon the left lower eyelid. On enquiry I found that Mrs. A. frequently left her

baby (affected with molluscum) in charge of the family to which this child belonged. She was a favourite with the children who played with her.*

On proceeding to squeeze the tumour on the breast, the delicate and vascular integument covering it burst and liberated the contents 'en masse.' The molluscum was in fact enucleated. A little bleeding occurred, which, after the ruptured integument had been removed with scissors, and the base touched with nitrate of silver, at once ceased. I shall presently describe more in detail the characters of this little tumour.

Six months later I saw this child again, and found a small tumour under the chin, and a larger pedunculated one on the back of the neck. The first mollusca had disappeared. I learned that there was another case in the same house, the subject being a child in a family lodging there. This was—

Case III.

E. B., æt. 4½ years. Has had numerous clusters of mollusca on chin and neck for about a twelvemonth. Is a constant playmate of Case II. Removed some of the tumours and contents.

Case IV.

Another brother of A. A. (Case I.), æt. 4 years, was brought, at my request, with a molluscum on his upper lip which has been there for six months. This was seventeen months after I first saw the sister.

I have now given the particulars of four cases which came before me, and have shown that while these occurred in three distinct families, which I shall call A, B, and C, a clear history of the disease was obtained as having affected the children of a fourth family D. Now, B, C, and D lived in the same house, and the children of family D, though living elsewhere, were frequently associated as playmates with those of C. I have only to add that these cases furnish to my mind sufficiently convincing evidence in favour of the theory of contagion. I shall moreover adduce stronger testimony from various sources as I proceed.

Respecting the question of contagion in this disease, we find

* I may state that I visited these families at their homes, and verified these statements as far as possible.

there is variance of opinion amongst writers on cutaneous medicine. Thus Mr. Erasmus Wilson states:* 'Of the non-contagious nature of these tumours there cannot be a second opinion.' And again:† 'We hold to the non-contagious side of the question, and believe in their endemic and epidemic nature rather than the capability of their communication by contact.'

M. Caillault‡ remarks: 'The attentive and continued study of many cases of molluscoid acne which existed simultaneously in different wards of the Hospital for Children, compels us now to believe in the possible contagion of this disease, and induces us to retract our former opinion that the epithet of Bateman was erroneous.'

Hebra§ observes: 'Some (English) writers believe that it is, and others that it is not, contagious. Of these opinions the latter is the one I myself hold.'

M. Hardy‖ believes the contagious elements reside in the spores of a certain cryptogamic parasitical plant. I believe no one has confirmed his observations, and I have never seen any such structures in the tumours or their secretions that I have examined.

Dr. Tilbury Fox¶ found in the sac of a tumour of molluscum a species of acarus, and he asks, 'Can the peculiar cells be the eggs of any animal parasites?' He remarks further, 'It is probable there are two varieties' (of molluscum). The cases he has seen lead him to think the disease may be contagious.

Dr. Neligan** remarks: 'I have no doubt in my mind as to its possessing the property of being propagated by direct contact, as from persons sleeping together, or in the case of children while at play.'

Mr. Jonathan Hutchinson†† has informed me that he recognises a contagious form of molluscum as distinct from a fibrous form met with more rarely in adults.

Dr. McCall Anderson of Glasgow informs me that he fully subscribes to the existence of a contagious form of molluscum.

* 'Diseases of the Skin,' 5th ed. p. 653.

† 'Student's Handbook of Diseases of the Skin,' vol. ii.

‡ 'A Practical Treatise on Diseases of the Skin in Children,' p. 75. Transl. Blake. 1861.

§ 'On Diseases of the Skin.' Transl. C. H. Fagge. 'New Syd. Soc.,' vol. i. p. 134.

‖ 'Leçons sur les Maladies de la Peau' (deuxième partie), p. 96.

¶ 'Skin Diseases,' p. 225. 1864.

** 'Practical Treatise on Diseases of the Skin,' 2nd ed. p. 305. Belcher, Dublin. 1866.

†† Quoted also by Fagge in his edition of Hebra, loc. cit.

Dr. Hillier * thinks 'the question of contagion must be held to be undecided.'

Dr. R. Payne Cotton † remarks that the disease may or may not be contagious, according to the character of the morbid elements which give rise to it, and these may in some cases be simply indurated sebaceous matter, or in others organised products of a different nature.

Dr. Craigie ‡ was amongst the earliest pathologists to recognise two forms of molluscum, the one contagious and the other not.

Professor Henderson § could not satisfy himself as to contagion in some cases he has very ably described.

Virchow ‖ believes that if the secretion of contagious molluscum reaches the sebaceous glands, new mollusca may be formed at the affected points.

I think it possible to reconcile these opinions by reference to exact and ascertained facts. In the meantime the testimony of the majority of writers is in favour of the contagion view. Confusion has no doubt arisen from the fact of there being two distinct diseases named molluscum. Mr. Erasmus Wilson, for instance, does not appear to recognise two different affections in M. contagiosum and M. simplex v. chronicum. Both forms have been carefully described, but I suppose it has but rarely fallen to the lot of observers to meet with cases illustrating them. The cases which I have collected and subjoined will, I believe, convey proof to the most sceptical non-contagionists that a distinct disease occurs which, under certain unascertained conditions, passes from child to mother, and so on through other members of a family or household. With the non-contagious form—a disease of adult life, and a grave one—we are no further concerned than to sufficiently depict its characteristics for the purposes of differential diagnosis. These will be shown later on.

I shall now exhibit in a concise form the history of a large number of cases which I have collected from various sources. Doubtless additional time and labour would have enabled me to add considerably to the list.

* 'Handbook of Skin Diseases,' p. 192. 1865.

† Report of some cases of Molluscum contagiosum. 'Edin. Med. and Surg. Journ.,' vol. lxix. p. 82. 1848.

‡ Description of a case of the Molluscum of Dr. Willan. 'Edin. Med. and Surg. Journ.,' vol. lxxv. p. 106. 1851. (This paper was written in 1819.)

§ 'Edin. Med. and Surg. Journ.,' vol. lvi. p. 213. 1841.

‖ 'Die krankhaften Geschwülste,' p. 221. Berlin, 1863. 1. Band.

*Dr. Bateman's Cases.**

Nurse caught it from child she tended. Two children in same family also suffered. In another family, an infant took the disease from a child who 'nursed' it.

Professor Thomson's Cases.†

In one family, the eldest boy was affected, supposed to have caught the disease from a playfellow. A younger sister and infant brother were subsequently affected. Mother who nursed the infant did not suffer. In a farmer's family a child was believed to have caught molluscum from a child of one of the farm-servants. Nurse of the child was affected on the neck where the infant usually laid its head.

Professor Henderson's Cases.‡

Three children in one family affected in succession. A neighbour's child in habit of associating with above. An orphan boy who had lived in a workhouse.

Dr. Paterson's Cases.§

Child of a Newhaven fishwife affected about mouth and nose. Mother suckled it from one breast only and became the subject of the disease, which was confined to the mamillary sebaceous glands. In another family a child was affected on neck and shoulders, and the disease was believed to be caught from a girl who carried it, and was the subject of certain cutaneous tumours.

The remaining two cases reported by this physician, I agree with Mr. Erasmus Wilson in considering unsatisfactory.

Dr. Neligan's Cases.‖

'I have myself witnessed two instances in which the acute form of this affection was communicated by children to adults, in each case members of the same family.'

* 'Delineations of Cutaneous Diseases.' London, 1817.

† Quoted by Paterson in his paper. 'Edin. Med. and Surg. Journ.,' vol. lvi. p. 280.

‡ Op. cit.

§ 'Edin. Med. and Surg. Journ.,' vol. lvi. p. 279. 1842.

‖ Op. cit. p. 305. By the acute form is signified M. contagiosum.

*Mr. Erasmus Wilson's Cases.**

A girl, æt. 4 years, had fifteen or twenty tumours. She recovered in a few weeks without treatment. Next, an infant sister and another, æt. 6 years, were brought suffering similarly; the mother also had several mollusca on her neck, and the scars of some that had lately healed on her face. After some months the disease occurred in some of the Meibomian glands in one of the children. Mr. Wilson considered this family to be of a sebaceous constitution.

M. Caillault's Cases.†

L. J., scrofulous girl, æt. 7. Numerous tumours on face, eyelids, and shoulder. In same ward, fourteen out of thirty children were affected with the same disease. M. Caillault noted that the first child affected was the youngest, and was often attended to by L. J., who in consequence of attacks of ophthalmia was much confined to the ward. He watched the cases with much care and was able to affirm that no new examples entered the ward.

An isolated case (E. A., æt. 7½ years) occurred in another ward containing scrofulous children. The occupant of the next bed (æt. 12 years) who was kept on her back with white swelling, and consequently attended upon by E. A., had at the same time (either from coincidence or contagion) a tumour of molluscum on the right cheek. The next case seems less doubtful.

Madlle. F. X., æt. 14, had for nearly six months thirty tubercles of molluscum on her neck and face. Her brother, æt. 18, had had on his face for a longer time than his sister some tumours exactly similar to hers. The family believed them to be warts, and thought they were communicated by means of towels which the brother and sister used in common.‡

M. Hardy's Case.§

A nurse had four or five mollusca on her breast; the child suckled by her had similar tumours on the parts of the face which came in contact with the diseased breast.

* Op. cit. p. 641. † Op. cit. p. 77.

‡ It is right to mention that M. Caillault reports some thirty cases of the disease, in hospital and other practice, in which he could not trace any contagion.

§ Op. cit. p. 98, and quoted by Blake in his trans. of Caillault.

*Dr. Addison's Cases.**

In some instances of the disease which came under the care of the late Dr. Addison at Guy's Hospital, and from which several wax models in the Museum there were copied, there was distinct history of communication of the affection from a nurse to children, and from the latter to other children.

Dr. Cotton's Cases.†

A girl, æt. 14, had the disease on hands and arms. Next, an infant sister, six months old, suffered on arms, chest, and thighs. Three months subsequently another sister had it on the head. The mother recollected having had something of the same kind when a child. The father, three other children, and some young neighbours constantly associating with those affected, all escaped. Dr. Cotton thinks the children may have derived some hereditary peculiarity from their mother.

Pathological Anatomy of Molluscum Contagiosum.

I shall first describe the characters of the secretion from these tumours.

If the scab-like punctum or hilum, as it may be termed, always met with on the summit of these growths, be removed with the point of a knife, and the tumour be firmly squeezed, a thin column of white pultaceous matter is caused to exude. It is not unlike newly-churned butter, and of the same consistence; it can be rubbed down between the fingers, and does not communicate a greasy stain to paper. It dries up into a starch-like cake. On microscopic examination the secretion is seen to be made up of numerous corpuscles, which are clearly referable to the epithelial type, and indeed most exactly resemble that variety known as tesselated.

Fig. 1.

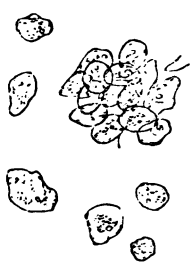


Fig. 1 illustrates their characters. On adding solution of caustic soda they swell up and become more distinct. The younger cells are smaller and more round;

* Catalogue, Guy's Hospital Museum. For permission to introduce these cases here, I am indebted to my friend Dr. Moxon, the Curator of that Museum.

† Op. cit.

some are oval. Finely granular contents are visible, most numerous in the younger cells, and in many of them nuclei are apparent.

*Description of Enucleated Mass from a Tumour.**

The following observations refer mainly to the examination of the small molluscum which was enucleated in my second case. The note I made at the time describes the naked eye characters thus—small mass, size of pea, made up of many small lobules, invested by a delicate vascular membrane. On the anterior aspect, corresponding to a central depression, is an aperture leading into a cavity. The appearance of the entire tumour somewhat resembles a small white raspberry, from which the calyx and thalamus have been removed.

Through the kindness of Mr. Bowater Vernon I have had the opportunity of examining another of these tumours, which he removed, enucleated and entire, from the lower eyelid of a young lad. It had been in chromic acid solution for some time and still contained some of the pultaceous secretion in its cavity.

These tumours, as seen removed from the cuticular cyst, resemble externally small racemose glands, such, for instance, as the salivary, the appearance of small lobules being very distinct.

In some instances it is impossible to remove the little tumour from within the cutaneous investment. In an early stage they are dome-shaped, completely sessile, and often rising from a broad base. In this condition, however, it is generally possible to cause extrusion of pappy contents by pressure after scraping off the epidermis, and this sometimes occurs during ordinary friction with a towel. The tumours may retain their dome shape without becoming pedunculated, and may also enlarge considerably, discharging from time to time. I am inclined to think that the occurrence of occasional discharge prevents the tendency to pedunculate in individual instances.

Microscopic examination of their sections illustrates the lobular and other arrangements, and I have figured the appearances disclosed on subjection of such an one to a power of 100 diam. linear (fig. 2). There is seen externally a somewhat delicate membranous investing sheath (*a*). Immediately within this lies the most dense and firm portion of the growth,

* Mr. E. Wilson, Prof. Henderson, and M. Caillault have all given good accounts of these tumours.

which requires a higher magnifying power to exhibit its intimate structure (*b*).

It is much paler than the internal part (*c*), the nature of which is, however, clearly shown. There is at once noticed the compound glandular character of the tumour, the lobules, as they may be termed, appearing mapped out by involutions of the fibrous investment. As just stated, the inner portion is alone clearly demonstrated by this magnifying power, and it consists of innumerable cells which are shed into the free central cavity of the little mass. It is quite obvious that this matter constitutes the pap which can be squeezed through the central aperture while the tumour is in situ, and of course after removal from the body. The cells are seen to be larger the nearer they approach the free surface, or, more strictly, the centre of the growth (*d*). They appear dark as viewed densely packed together. I should state that this preparation was examined first in the recent state, and subsequently with the addition of glycerine.

Fig. 2.

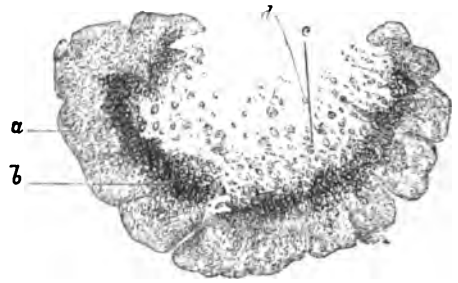


Fig. 3.

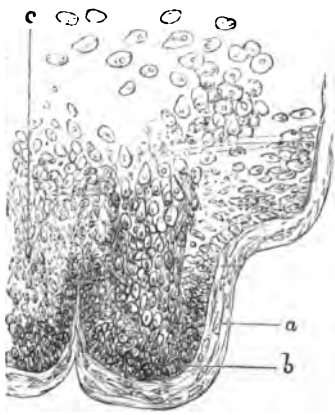


Fig. 3 illustrates the more minute structure of the dense peripheral portion magnified 320 diameters linear. At *a* are seen the nuclei in the fibres of investment. Myriads of closely-packed cells abut upon this membrane, placed side by side and vertically for the most part (*b*). The addition of glycerine renders their nature and arrangement more clear. The same appearances are seen when the section has been previously steeped in chromic acid solution. Passing inwards the cells are observed to be larger till they reach their maximum size most internally as seen at *c*.

Of the cuticular covering of these tumours I have nothing

particular to remark. In many of them may be seen small ramifying bloodvessels, and the dermal membrane is generally so delicate that the contained tumour gives a pearly aspect to the mass.

Having thus described the class of molluscum to which, as I believe, has rightly been assigned the epithet 'contagiosum,' I may remark that other morbid conditions have received the same name, or have been classified as different forms of molluscum. Several writers have subdivided the contagious form into varieties, these being taken from the naked eye characters, and there can be no objection to this so long as a distinct disease is not included in the category. This leads me, however, to discuss the

Pathology of Molluscum Contagiosum.

I believe we shall do rightly if we adopt the teaching of such pathologists as Craigie, Gustav Simon, Hebra, and Virchow, in regard to this affection. Thus, according to Gustav Simon,* there are two kinds of molluscum, one M. 'simplex,' the other M. 'contagiosum,' the former consisting of fibrocellular outgrowths unconnected with follicular structures, the latter being due to increased growth in sebaceous glands.† Dr. Beale‡ has described with great minuteness a case of molluscum. Careful perusal of his report at once enables us to refer this instance to the variety 'simplex' (Simon) v. 'chronicum' (Craigie). This disease has been well described by these writers,§ as well as by Hebra and Virchow. It must be regarded as entirely distinct. The famous case reported by Professor Tilesius‖ was no doubt of this nature, and it is believed to be the first accurately recorded. It seems to be peculiar to adults, and is said to be more common in males. There is no history of contagion, and the disease occurs on all parts of the body. The tumours are apt to enlarge considerably, which they never do in the contagious form of molluscum. Again, the general health is affected in these cases, while this is never so in instances of the minor affection, though

* 'Die Hautkrankheiten,' pp. 235 and 354. Berlin, 1851.

† Vide Paget's 'Lectures on Surgical Pathology,' note on p. 452. 2nd ed. Turner.

‡ 'Path. Soc. Trans.,' vol. vi. p. 313. § Op. cit.

‖ Vide Craigie's admirable analysis of Jacobovic's treatise, op. cit. p. 115; and Virchow, op. cit. p. 325.

there may perchance be a strumous habit coexisting (Craigie, Fuchs).

Beale's examination of Mr. Bowman's case led him to believe that the tumours consisted essentially of a morbid alteration in the structures concerned in the formation of the hair, especially in the cells at the deepest part of the follicle, and in the follicle itself, and that the subcutaneous areolar tissue was considerably hypertrophied while the sudoriferous and sebaceous structures were unconcerned in the growths. He found also what he conceived to be altered hair structures in the centre of several tumours. Now, it is clear that there is an essential difference between these growths and those of the contagious kind. I have not met with any structures resembling hair in the small mollusca from children, nor have I observed hairs growing out of these tumours, though I have seen them in one case of fibroma molluscum. I fully agree with Hebra,* who remarks: 'If we are to term this affection "molluscum," we may distinguish two varieties of it by naming the one M. fibrosum, or, as Virchow has it, "Fibroma molluscum," and the other "M. sebaceum," s. "contagiosum."'†

* Op. cit. Vide plates illustrating the two affections. Willan was one of the earliest English writers on the chronic form.

† Thanks to the kindness of my friend Dr. Hilton Fagge, since writing the above, I have been enabled to see two cases of the fibrous form of molluscum. The first was from amongst his out-patients at Guy's Hospital—a woman, æt. 34. She has had numerous tumours for about ten years. They came gradually, and vary in size from that of a pin's head to a walnut. Some of them are pigmented, much like the so-called sultana raisins in appearance, pedunculate also and pendulous. They possess no scab-like stigmata, nor can any contents be forced out of them. Others, and especially the larger ones, are doughy to the feel, the skin covering them appearing natural in texture though possessing a peculiar mauve tint. They are numerous on the face, shoulders, and mammæ. The largest is over the left hip. The tumours are painless, and insensible to pinching. Hairs protrude from some of them. The general health of this woman is excellent. She has been married nine years, and has suckled five children without once communicating the affection. There is no history of such a disease amongst her immediate relatives.

The second case had been in Guy's Hospital and also in the London Hospital, for the relief of certain tumours on the right leg. I went to her home in Essex to examine her condition. Her age is 22, she is single, and has lived in a miserable hovel all her life. She has numerous tubercles of fibrous molluscum on various parts of the body. These commenced to grow when she was about fourteen or fifteen years of age, and new ones constantly appear. At first they are not larger than a pin's head or millet seed, and gradually grow till they assume the proportions of a pea or in some cases even of an orange. They are abundant over the front of the breast. Both nipples have been enlarged for four or five years, and are now firm and fibrous to the feel. Several pendent portions are attached to them. There is a tumour near the left mamma the size of a walnut, tender when pinched. It is of a mauve tint at the base, and becomes browner towards the free extremity. As in the first case, there are several of the purplish cushiony tumours of various sizes amongst the groups, and in some instances the skin is commencing

Virchow* has expressed himself as follows:—'Ein genetischer Unterschied zwischen Molluscum contagiosum und non-contagiosum ist jedenfalls nicht bekannt; man könnte höchstens die mit geschlossenen Ausführungsgängen für nicht-contagiös und die mit offenen Ausführungsgängen für contagiös erklären.' This statement is supported by the known facts of the two affections.

In the present state of our knowledge, I believe we approach nearest the truth if we describe these tumours (M. contagiosum) as diseased conditions of the sebaceous glands. The teachings of direct inoculation having for the present failed us, we are nevertheless, I consider, entitled to suppose that the secretion of mollusca is the most likely element in propagating them. Bateman and others believed this. The individual cells probably possess the inherent power of exciting in the healthy sebaceous cell an abnormal stimulus, whereby its activity is increased and its properties altered. The secretion reaches these glands through their openings, and the minute hairs may serve to retain and conduct it. The largest tumours, I believe, are the result of this morbid action set up in the compound sebaceous follicles, the individual sacculi becoming hypertrophied, thus forming the botryoidal or racemose appearance in the entire tumour. The active centres of growth are doubtless the lining layers of glandular epithelium, which are con-

to shrivel over them. On certain of them were situated two or three small brown tumours, and from more than one I was able to squeeze by firm pressure a very little whitish fluid. From another I removed with my thumb nails a mould of a distended hair follicle with a blackened head, such exactly as one would term the contents of a specimen of acne punctata. None of the tumours possessed the stigmata of M. contagiosum, nor did I observe any emergent hairs. I explain the facts just mentioned by supposing that so extensive a disease could not fail to involve numerous hair and sebaceous follicles, and in some instances to irritate them considerably. There were numerous tumours in the grooves of the back and upon the arms. The largest growths were upon the right leg; one the size of an orange had been removed from over the lower third of the tibia, and a smaller one from the neighbourhood of the internal malleolus. The integument over the lower third of the limb was hard and brawny, but no smaller tumours were found here or on the other limb, neither did any exist on the lower part of the trunk. The growths on the face were small and flattened, more or less white, not brown, mauve, or pedunculated. I could not fail to be reminded on seeing this poor girl of the remarkable instance of the disease which Virchow has placed on record, and likewise had engraved for a frontispiece to his great work on tumours,[1] though there were certainly no growths of such size as he there depicts. The general health of this patient was indifferent. She complained of weakness and incapacity for her household duties, and this has increased during the last year. No other members of her family were similarly affected.

* Op. cit. 1. Band, Follicular-Cysten, p. 223.

[1] Vol. i. op. cit.

tinuous with the mucous layer of the skin. The morbid impulse conveyed here is followed by active cell-formation, great enlargement of the entire gland, and its upward extrusion, in the manner so well described by Wilson,* above the level of the skin. Its advance in this direction, indeed, is entailed by its anatomical position. The cuticle advances before it, becomes thinner and thinner by pressure, while the so-called hilum or scab, which is always seen towards the summit, remains to show, according to Hardy, the original orifice of the obliterated follicle.

As already stated, we are ignorant of the conditions under which the affection proceeds. I believe the disease is unknown amongst classes of individuals who follow the common laws of hygiene. Doubtless dirt, overcrowding, and the unhealthy conditions inseparable from poverty, are important factors in the propagation of the disease.

I think we may fairly assume that contact is necessary to originate the morbid process, and it is at least possible, as both Mr. Wilson and Dr. Cotton suggest, that certain persons may possess a sebaceous constitution or diathesis, with special tendency to diseases of this portion of the cutaneous system.

Natural Tendency of the Disease.

Several modes of termination have been observed. Sometimes the little tumours drop off spontaneously, or an inflammatory process arises which leads to what may be called a radical cure. Again, they may become pedunculated, in which case they tend to drop off.

An unsightly ulceration also occurs in the summits of the tumours, and the contents are thus shed, and in fact slough or suppurate out. I have seen ugly cicatrices as the result of this. A very noteworthy removal of the disease, unique so far as I know, was effected in the case of a little girl under the care of Dr. Church, who suffered from scarlatina. During recovery from the fever all the mollusca came away with the desquamating cuticle.

The affection may last from a few months to a year and a half, unless interfered with by treatment. The tumours are remarkably painless, and bear any operation. I need not here allude to the further contrast between this disease and fibrous molluscum, which is brought out by a study of the natural mode of cure in the milder affection.

* 'Student's Handbook,' vol. ii.

Inoculation Experiments.

These appear to have failed in every instance. Attempts were made by Professor Henderson and Dr. Paterson to propagate the disease in this manner. Hebra likewise inoculated but without success. Zeissl, quoted by Fagge, denies the contagiousness of molluscum, because his, and the experiments of Hebra and others on inoculation failed. He had, however, never seen the disease in children. As Dr. Fagge very rightly remarks, if the mollusca in these cases were merely the sessile or pedunculated tumours of adults, the failure of these experiments can hardly be considered to disprove the possibility of contagiousness.

In conclusion, I would remark that there is a popular belief that ordinary warts (verrucæ) are contagious. I am not aware that there are any well authenticated cases on record to prove this. It has occurred to me that this opinion may perhaps have had a partial origin in the facts that have been so frequently observed in cases of molluscum contagiosum, and where, it would appear, the evidence in favour of contagion, under certain conditions, is incontestable. It is not unlikely that these little tumours may have been mistaken by the ignorant for true warts.

ARTICLE XIV.—*Case of Elephantiasis Arabum successfully treated by Ligature of the Superficial Femoral Artery.* By T. SYMPSON.

FOR notes of the following case I am indebted to Mr. Samuel Mills, House Surgeon to the Lincoln County Hospital.

L. A., æt. 41, unmarried, an union schoolmistress for twenty-one years, was first admitted into the hospital on December 23, 1867, suffering from elephantiasis of her left leg and foot. The enlargement and disease of the limb were at this time limited to the parts below the knee, the tissues of the thigh being scarcely, if at all, infiltrated.

She states that for thirty-one years her left foot and leg have been larger and more vascular than the right, but it is sixteen years since her leg began to swell and become materially diseased and painful. Within that period it has varied more or less in size, sometimes becoming extremely

painful and irksome to her, and thus rendering her unfit for the performance of her duties until the inflammation had subsided.

On December 24, 1867, the leg was in the following state: The limb, from just below the knee to the ends of the toes, was very much swollen, apparently from hypertrophy of the skin and subjacent soft parts, whereby its natural contour was obliterated and its symmetry effaced, the thickest and most prominent parts being over the instep and just above the ankle, where the enlarged and altered tissues formed great bosses divided by a deep sulcus. The surface was arranged into transverse corrugated lines with intervening furrows, and the natural cuticular surface appeared lost and replaced by rough irregular scales of a reddish-brown tint. The front aspect of the lower half of the leg was occupied by a large irregular patch of tubercular-like ulceration. The granulations being very vascular, prominent, and bright scarlet in colour, gave to this part a raspberry-like appearance and arrangement. This kind of ulceration affected also the dorsum and the sole of the foot in irregular circular patches, and, wherever present, it yielded a very offensive thin discharge. The three middle toes were matted together, and formed one large fungous mass ulcerated upon the surface. The great toe, although separate, was distorted, and was affected by the same kind of hypertrophy and of ulceration. All the ulcerated parts were excessively vascular and highly sensitive. The tumefaction and engorgement of the limb were always much increased by her being up and about.

The following comparative measurements of the legs were taken after she had been lying quietly in bed for a few days:—

	Healthy limb (right)	Diseased limb (left)
Around the calf . .	12½ inches	15 inches
Above the ankle . .	10¼ „	14 „
Across the instep . .	9¼ „	12¼ „

The right leg, from the ankle to about four inches above it, was to a slight extent the seat of hypertrophy of the skin and subjacent connective tissue, and was marked by three cicatrices —a large one extending half-way round the back of the leg just above the ankle, and two others situated somewhat higher on the anterior and external aspect of the limb. She stated that, twenty-five years ago, after a severe attack of inflammation, large 'cores' came from the situations now occupied by the cicatrices. About the same time her right cheek became in-

flamed and a portion of the malar bone came away. The malar bone, zygoma, and adjacent tissues remained thickened.

She had not, to her recollection, suffered sprain of either limb, nor had she had intermittent fever.

Her general health had never been very good; when a girl she passed favourably through an attack of scarlet fever; but for many years she had been subject to inflammation of the left leg, recurring at intervals of from three to five weeks sometimes for two years together, afterwards ceasing for a year or two, attended with considerable constitutional disturbance, and on each occasion leaving the limb more greatly enlarged and indurated. The attacks commenced with nausea and vomiting, which were followed by extreme tension and swelling, as well as excessive pain of the limb, which was of a scarlet redness. On the week succeeding the cessation of the attack desquamation of the cuticle always occurred. She first noticed the occurrence of any thickening in the form of a small puffy nodule on the second toe.

Her mother died, when 35 years of age, of phthisis; her father, at 29, of some complaint in his back. She had one sister living, 44 years old.

The leg was dressed with a weak solution of carbolic acid (1 to 30), and she was ordered a mixture of tincture of perchloride of iron and quinine.

On January 1, 1868, local pressure was tried by means of tight bandaging, but this had to be left off on account of the excessive tenderness of the granulations.

21.—Iodide of potassium in scruple doses was given in a bitter infusion three times a day.

25.—Iodism produced. To omit the iodide of potassium.

February 20.—Pressure on femoral artery exerted by means of a Skey's tourniquet, which was worn for about an hour every day. This was kept up more or less for a month.

March 23.—The limb considerably improved in many respects, being altogether diminished in bulk and the skin being looser. At her own request she went out relieved.

30.—The patient, after being out a week, returned, and sought readmission into the Hospital on account of the leg getting much worse. She was now willing and almost anxious to have it amputated, or to submit to any other operative measure that might be deemed advisable. The swelling and engorgement of the foot and leg were very considerably increased, and the pain and excessive tenderness of the ulcerated parts were such as to render her existence miserable. She

was accordingly admitted, ordered to remain quiet in bed, and the leg dressed with water dressing (carbolated).

April 3.—The urine on examination was found to be turbid from deposit of lithates, free from albumen, and having a specific gravity of 1023.

8.—Pressure on femoral artery by tourniquet again tried. Ordered to take cod liver oil and syrup of the iodide of iron.

27.—Leg not so well again. There was a scarlet blush on front of the leg and over the knee. She had had slight rigors and was feverish. Ordered to take citrate of potash in the effervescing form; hot fomentations to be applied to the limb.

May 2.—The erysipelatous blush had disappeared. To resume the cod liver oil and iodide of iron.

26.—As the limb was at its best simply a painful encumbrance to her, and as the disease appeared to have been almost wholly uninfluenced by the treatment hitherto adopted, it was proposed to place a ligature on the main artery of the limb, to which she readily consented. She was at this time in pretty good health, the bowels regular, and the urine free from albumen, of sp. gr. 1030, and merely turbid with lithates.

28.—11 A.M. Chloroform having been administered, Mr. Sympson ligatured the superficial femoral artery at the apex of Scarpa's triangle at about four inches from Poupart's ligament. No difficulty was experienced during the operation. In passing the needle the vein was carefully avoided, and on its withdrawal, armed with strong whipcord, the internal saphenous nerve was held outwards by forceps until the ligature was tightened. The lips of the wound were approximated by three silver sutures and intervening broad strips of plaster, a light pad of lint and a bandage being applied over the whole. The limb from the toes upwards was then enveloped in cotton-wool, kept in position by flannel bandages. The patient seemed remarkably well after the operation was completed.—2 P.M. Complained of gnawing pain about the inner part of the knee and also in the lower part of the back. Had half a grain of acetate of morphia in solution given at once. The toes retained their former colour, but were not quite so warm as those of the opposite limb.—8.30 P.M. Had been much easier since she took the sedative, but still felt slight pain about the inner part of the knee and leg. The temperature of the foot, as judged by the sensation conveyed to the hand, had increased, and she said that the limb felt warm. To have a second sedative draught ready, to take in the night if required.

29.—Had passed a tolerably good night, and did not require

the second sedative draught. She still complained of a 'wearying' pain in the lower part of the back. Pulse 72. No constitutional disturbance. The bandages and dressings were not disturbed. A thermometer slipped under the foot, between it and the cotton wool gave as the temperature of the diseased limb 97·2° F., that of the healthy limb being 97·6° F.

30.—Dressings not disturbed. Temperature, diseased limb 96·2° F., healthy limb 94° F.

31.—Temperature of diseased limb taken in the evening was 98° F. The wound in the thigh was dressed this morning and was looking well; two of the sutures were snipped through and removed; some union had occurred at each end of the wound, but there was a small amount of suppuration in the track of the ligature. On examination of the limb it presented a very marked difference to its appearance before the operation, the ulcerations being far less prominent and less vascular, and the general bulk of the leg and foot from the knee downwards having visibly decreased.

MEASUREMENTS OF THE LIMB.

	Before the operation	On the fourth day after the operation
Above knee	14¼ inches	14 inches
Middle of calf	14½ „	14 „
Above ankle	14 „	13 „
Across instep	12¼ „	11 „

The ulcerated parts were dressed with dry lint, and the limb again enveloped in flannel bandages.

June 1.—Complained of aching pain in the back and knee. Pulse quiet. Temperature of diseased limb 97° F.

2.—Temperature of limb 97·4° F.

4.—Temperature of limb 98° F.

6.—The following measurements were taken to-day:—

Above knee	14 inches
Middle of calf	13 „
Above ankle	12½ „
Across instep	11 „

11.—The previously matted-together toes were this morning separated by placing between each toe narrow strips of lint, the sores on the leg and dorsum of the foot were covered with dry lint, and a calico bandage, rather tightly applied, was substituted for the flannel one. The temperature of the limb seemed about normal. The wound in the thigh was suppurating slightly, to it was applied water-dressing retained in position by a light bandage.

17 (twenty-first day after the operation).—The femoral ligature separated this morning; it was removed without any dragging and without the show even of a drop of blood from the granulations. The patient was in a good state of health and had lost the pain in the knee and lower part of the back. She took the ordinary diet of the hospital and a pint of beer daily.

The dimensions of the limb were again taken:—

Above knee	14 inches
Middle of calf	12½ „
Above ankle	12⅛ „
Across instep	11 „

July 11.—The ulcerations were healing slowly, the skin and subcutaneous tissues were much more supple, and the bosses about the ankle had become flattened, so that the outline of the joint was now visible, and she could move it with ease. The leg was simply dressed with lead lotion, and the bandage discontinued.

22.—The wound in the thigh had quite healed; the sores on the leg were gradually improving.

August 22.—She had continued to improve since the last note; was now up the greater part of the day, walking well with a scarcely perceptible limp. The contour of the limb much improved, the skin nearly natural in appearance, the ulcerations almost healed. She could flex and extend the foot with ease, and had in great measure regained power over the toes.

The limb afforded the following measurements:—

Above knee	13½ inches
Middle of calf	12¾ „
Above ankle	12¾ „
Across instep	11 „

The bandage was again applied.

In a disease so little amenable to the employment of remedies as elephantiasis arabum has hitherto proved it seems extremely desirable that any evidence, confirmatory or otherwise, of the efficacy of a new mode of treatment should be placed upon record, more especially as the rarity of the occurrence of examples of the complaint in this country must necessarily for some time prove an obstacle to the practical testing of any method of attempting to arrest its progress.

The foregoing case tends forcibly to demonstrate the value of the plan of treatment devised by Carnochan and successfully carried out by him, as well as by Statham, Butcher, Alcock, Bryant, and Buchanan.

The issue of these cases having been so extremely satisfactory makes the want of success of Professor Fayrer in two instances in which he tied the main artery of the limb appear the more remarkable, until we recollect that the best authorities agree in looking upon the disease as one very largely influenced by locality. Mr. Nayler, who has had great experience in India, tells us* 'that a removal to a distant station in India, unless the complaint be far advanced, is generally followed by a remission of the local symptoms. The swelling may even disappear, but a relapse is almost sure to arise on the patient's return to his former abode;' hence it seems reasonable to suppose that in regions where the disease is prevalent the characteristic lesions of the tissues would be more marked and intensified than where it is merely sporadic, and Mr. Erasmus Wilson† does not hesitate to attribute the failure of Professor Fayrer to the endemical character of the complaint, as well as to 'the probability of extension of disease from the vessels of the fibrous and cellular structures of the integument to those of the rest of the limb.'

Of the Professor's two cases, one died of pyæmia, the other derived but little benefit from the operation.

It is apparently impossible to assign a cause for the occurrence of the disease in my patient. None of her relatives had suffered in a similar manner. She had, when young, been attacked by scarlatina, but from this she had made a good recovery. She had never suffered from intermittent fever, nor had she at any time been placed under unfavourable hygienic conditions. She was a native of Leicester, but for many years lived in the neighbourhood of Great Yarmouth. She had never been abroad.

At first sight the case did not appear to be an encouraging one, for prior to entering the Hospital she had, under the care of numerous medical men, been subjected to a great variety of treatment with but trifling alleviation of her sufferings. Still, on consideration of the satisfactory termination of cases in which the circulation had been modified by placing a ligature on the main artery of a limb, as well as witnessing the marked beneficial effect in this case itself of rest and compression of the femoral artery, I came to entertain the hope that ligature of this vessel might prove successful in curing the disease, and h us render amputation of the limb needless. It is extremely

* 'Treatise on Diseases of the Skin,' p. 254.
† 'Journal of Cutaneous Medicine,' vol. i. p. 187.

satisfactory to find one's anticipations in a fair way of being realised.

As to the name of the disease, I have ventured, like Dr. McCall Anderson,* to call it elephantiasis arabum, not because I am insensible of the force of Mr. Erasmus Wilson's objections to that term, but from its being more commonly in use, and, at present, probably more likely to be understood than its synonyms 'spargosis' or 'bouknemia.'

Article XV.—*Miscellaneous Ophthalmic Cases.*

By Bowater J. Vernon.

THE following cases, though briefly narrated, are of sufficient interest to be put upon record. The dependance of various affections of the eye and its appendages upon morbid conditions of the jaws and teeth, is a well recognised clinical fact, and in the present instance the connection between them, as cause and effect, seems to have been indisputably shown. It is probable that the immediate cause of the protrusion of the eye was some serous effusion within Tenon's capsule; and Mr. O'Ferrall† has shown how a comparatively slight amount of effusion in this situation may cause a very considerable displacement of the globe.

I can find no case of injury to the head on record where the sixth pair of nerves only has been damaged and on each side at the same time: from the anatomical relations of the parts, it is possible to imagine a fracture running through the body of the sphenoid bone in such a direction as to involve the trunk of the sixth nerve and to open into the cavernous sinus without interfering with the other nerves in the vicinity, provided there was no material displacement of parts; and the fact of the sixth nerve on the left side entirely, and the opposite nerve to some extent recovering their power, leads to the supposition that their trunks were stretched or pressed upon by hæmorrhage rather than actually torn across. The double convergent squint was a striking disfigurement, and for some time the vision was extremely confused by double images of objects; there was some difficulty in determining the exact position of these images, as the patient's answers could not always be relied

* 'Journal of Cutaneous Medicine,' vol. i. p. 183.

† 'Dublin Medical Journal.'

upon; after a time, however, they ceased to be a source of annoyance. Repeated examination with coloured glasses failed to detect any want of power in any of the muscles of the eyes, except the external recti.

The three cases* in which amaurosis seemed to be caused by pressure upon the globe or optic nerve from without, by congestion of the vessels or effusion and inflammatory changes taking place in the soft tissues of the orbit, seem to point out the advisability of active treatment, such as incisions, in those cases where the eyes remain closed by extreme swelling of the eyelids. The last of the three cases, and the most recent under observation, is of great interest, inasmuch as it is easy to read in the ophthalmoscopic appearances of the fundus the remarkable changes which have occurred in the circulation of the optic nerve, and the order in which they took place. It is easy to understand the distension of the retinal veins owing to the obstruction of the reflux of venous blood into the orbit, by the pressure around the optic nerve, and as that pressure increased the veins would be distended and would give way, and at the same time no arterial blood could flow through the arteries, so that their position could only be recognised by their appearance as so many minute white threads; considering the time which must have elapsed since the sight was completely lost, it is improbable in this instance that any perception of light can ever be restored, but in similar cases there is good ground for believing that if measures can be taken to relieve the pressure upon the optic nerve, or to prevent its being stretched by the protrusion of the eyeball, the nerve fibres may be saved from complete disorganisation, and that in time at least an useful amount of vision may be regained.

Case I.—*Protrusion of the eyeball removed by the extraction of a decayed tooth.*

Henry Vine, æt. 17, came to the Hospital August 5, 1868, on account of considerable projection of his right eye, which had commenced about ten days before, and was still increasing. The eyeball was so prominent that the eyelids scarcely covered it; still there was no loss of muscular power, and its movements were not materially impaired. The conjunctiva was congested, and there was excessive lacrymation, but the eye itself seemed healthy and the acuteness of vision normal.

* The account of the first two of these cases is a brief abstract from notes taken when one of Dr. Burrows's clinical clerks.

After very careful examination, no growth or swelling could be detected behind the eye, but there was some pain on pressure with the fingers into the orbit. He had suffered a good deal lately from tooth-ache and neuralgic pains in the face and right temple, but in other respects had enjoyed good health, and had never met with any injury to his head or face. The muscles around the right eye occasionally twitched, and his right cheek seemed rather swelled. The teeth in the right upper jaw were perfect. In the right half of the lower jaw the first molar tooth was broken away by caries to the level of the gum, which was tender and much thickened on both sides.

As the irritation in the mouth seemed the only probable cause for the condition of the eye, the extraction of the offending stump was insisted upon and at once performed; and at the same time he was instructed to use frequent hot poppy fomentations to his face.

On Saturday, August 8, he came to express his great relief from the removal of the tooth: all his pains had left him, and the eye had almost receded into its natural position. In another week's time he seemed in every respect to be perfectly well.

Case II.—*Paralysis of the sixth nerve on each side, after an injury of the head.*

James Hollins, æt. 15, a printer's boy in Clerkenwell, was admitted into Colston Ward under Mr. Holden, on the evening of May 5, 1868; about half an hour before, his head had been crushed against the wall by some part of a printing press, which struck him just above the left ear. He was sensible but extremely pale, with widely dilated pupils, and a convergent squint when brought to the Hospital. The squint was noticed by those who picked him up, and had not existed before the accident.

Soon after his admission he lost a large quantity of dark bloody fluid from the left ear and the nostrils.

In three days' time, when fit to be more thoroughly examined, he was quite able to give an account of himself, although still very feeble and extremely pale. He was not at all deaf and was able to move his head without any inconvenience; there had been no further bleeding from the ear. He complained of pain in his temples, and of seeing everything double; both his eyes squinted inwards, the right more so than

the left, both pupils were dilated and sluggish, all power over the right external rectus muscle was lost, but some slight control still existed over the left external rectus, the double images were only noticed in the horizontal planes; the vision of both eyes when tested singly was quite normal.

During the next fortnight the squint on the right side seemed to increase while that on the left side decidedly diminished; about a month after the accident the squint on the left side had disappeared, and power over the external rectus seemed thoroughly restored; some power returned also over the right external rectus, but there still remained a squint of 4‴ in degree. There never appeared to be any loss of power over any other muscles, though several careful examinations were made. He remained in the Hospital until August 20: his health was restored and his colour had returned, but the squint on the right side remained permanent; he had had no trouble from double images for some time previously to his discharge.

Case III.—*Erysipelas of the face—Suppuration within the orbits—Amaurosis on the left side.*

James Collins, æt. 32, a bricklayer by trade, came under the care of Dr. Burrows in Matthew Ward, April 25, 1862. He had been ailing for some weeks, and had erysipelas of the head and face for a week before his admission. The general symptoms were very severe: extreme prostration with delirium, and for days he was hardly conscious.

The eyelids on both sides were exceedingly swelled, and profuse purulent discharge poured from beneath them; incisions were made into them on both sides; some weeks elapsed before he could open either of his eyes, and it was then found that the left eye was absolutely blind, being unable to distinguish light from darkness; the vision of the right eye was considerably impaired at first, but as his general condition improved this was completely restored. The left eye remained blind; the pupil was sluggish, rather dilated and irregular, and the whole globe seemed more prominent than its fellow, and restricted in its movements; during his convalescence a large quantity of pus made its way through the conjunctiva by the side of the eyeball.

Before leaving the Hospital he was again in extreme danger from typhus fever, but he was eventually discharged quite well, although blind on the left side.

CASE IV.—*Urticaria—Erysipelas of the face—Protrusion of the right eye from suppuration within the orbit—Temporary blindness.*

Rosina Myenski, æt. 17, a Jewess, in very bad health and circumstances, became a patient under the care of Dr. Burrows, in Hope Ward, July 18, 1861.

For some time she had been subject to attacks of illness which seemed to terminate in a copious eruption of urticaria. A week after her admission she was attacked by erysipelas of the head and face of the most severe character. For many days both eyes were completely closed, owing to the extreme swelling of the eyelids: on the right side a large collection of matter formed in the upper eyelid; several small incisions into this gave a temporary relief; these had to be extended into the orbit above the eyeball, and for days the discharge from them was profuse, accompanied by sloughs of cellular tissue of a considerable size. For three weeks this eye remained almost blind, though perception of light was still retained; as her recovery took place the vision became completely restored, but the movements of the eyeball were very limited in all directions, and vision for near objects was attended with difficulty. After leaving the Hospital in this condition no further note could be made of her progress.

CASE V.—*Erysipelas of the face—Suppuration within the right orbit—Complete amaurosis on this side.*

James McKenzie, æt. 72, struck his forehead against a gas-burner on June 3, 1868. In two days' time his forehead and face were so much swelled that his eyes were completely closed. He was admitted into Abernethy Ward, under Mr. Savory.

For a month he could not open either eye, suppuration occurred in the eyelids, and he suffered a good deal of pain in the eyes themselves. The left eye was opened about a fortnight before its fellow, and he could see fairly well with it; when the right eye was opened it was slightly prominent and divergent, and quite unable to distinguish light from darkness.

When I examined him on July 20, he was up and about the ward, and had regained his strength; the eyelids on each side were red, œdematous, and marked with scars; the right eye was more prominent than the left, the conjunctiva was œdematous and much thickened, and the movements of the globe restricted, the cornea was healthy, the iris rather discoloured,

and the pupil irregular and fixed, there was no perception of light remaining. Vision with the left eye was not impaired, and with his spectacles, which he had used for some years, he was able to read well.

The pupil of the blind eye, after some trouble, was dilated with atropine, and the fundus examined with the ophthalmoscope.

The crystalline lens was rather hazy, but the vitreous humour was quite clear, and the back of the eye could be clearly seen. The optic disc was very pale, and presented no appearance of any bloodvessels upon its surface; on its apparent lower part was a dark grey patch, which looked like the remains of an old hæmorrhage upon the surface of the disc; the retinal arteries could not be traced, but the veins were very much enlarged and varicose. I could find no traces of hæmorrhage, or of morbid change elsewhere.

After leaving the Hospital he attended under my care at the out-patient room. The loss of his vision has remained absolute; a collection of pus has made its way from the orbit on the inner side of the globe, and a mass of granulations has sprouted from the sinus thus formed.

On examination with the ophthalmoscope, just one month after the previous examination, the same appearances presented themselves, and in addition I could detect the position of the retinal arteries by a network of silvery white streaks which accompanied the dark and distended veins.

In connexion with this case it is a matter for regret that no ophthalmoscopic examination was made of the condition of the fundus in the two preceding cases.

Article XVI.—*Cases of Masked Hernia, and other cases.*

By George W. Callender.

We are familiar with cases of hernia, especially of the femoral variety, in which, from continued strangulation, the extremity of a loop of intestine becomes gangrenous. Whilst the wall of the gut most distant from the mesentery is the hernia, and is strangled and dies, the remainder of the wall, comprising often two-thirds of the circumference, remains within the abdomen, and, except by continuity of structure, forms no part of the rupture. It is in such cases (as dis-

tinguished from those in which an entire loop of the bowel, including its mesenteric attachment, escapes from the abdomen and becomes strangulated) that perforation of the intestine with discharge of fæces may be followed by closure of the fistula, and by complete recovery.

With fæcal fistula from gangrene of a strangulated rupture there are two results—(*a*) If an entire loop of bowel has perished, the fistula, if the patient survives, is final; the upper and lower openings in the bowel lie side by side, and never of themselves conjoin, nor can they, despite Dupuytren's suggestions, be made to unite by any surgical operation; a loop of intestine has been, as it were, cut out from the canal, the breach is permanent, and fæces will ever escape through the fistula. (*b*) If the extremity or only a small portion of a loop has mortified, the fæces pass through a comparatively minute opening in the bowel, and as the fistulous aperture cicatrizes the contents of the intestine flow less and less freely through the artificial anus, and at last cease to do so, finding their way along the natural canal, which is, however, narrowed, at the site of the fistula, to an extent corresponding precisely to the area of its wall lost in the progress of the gangrene. In such cases the fistula is slowly but eventually closed, to the great comfort of the patient.

All this is pretty well known, but it has, I believe, escaped notice that a hernia of the latter kind may exist, and may become strangulated, and that the patient may die without having any of the ordinary symptoms of the disease. In illustration of this I will relate some cases which, at intervals of time, have come under my notice.

A female, æt. 62, was admitted into St. Bartholomew's, said to have a strangulated hernia, and having suffered from attempts made to reduce it. There was a history of the hernia having suddenly descended, and of subsequent constipation and vomiting, but this was a week ago: since then the sickness had subsided, and the bowels had acted freely several times, nor was the patient suffering from aught save the pain of an abscess in the groin which threatened shortly to break through the skin. On opening this I let out a quantity of fœtid pus, having an intestinal odour, and gas. The opening failed to close, and intestinal contents passed through it so long as she remained under my observation. During this time the bowels continued to act freely and with regularity. It was not considered desirable in this case to attempt to close the fistula by operation. There was no doubt but that the extremity of a

loop of bowel had become gangrenous. The absence of symptoms was due to the patency of the canal behind the strangulated portion, whilst peritonitis was limited by adhesions formed behind and around the protruding gut.

Whilst I was house-surgeon to Mr. Stanley, we were sent for one day by Dr. Roupell, to see a case in Luke Ward, where we found a robust fresh-coloured lad, æt. 18, who had just arrived from the country, stating that six days previously he had been suddenly seized with pain in the abdomen, chiefly in the lower left side. The bowels had acted since, but he had felt sick, although he had not vomited. His pulse was 90, and his tongue was furred.

About the situation for a femoral hernia there lay an oval tumour, evidently an enlarged gland. The abdomen was swollen and tender, especially over its lower half. Beyond this evidence of stomach mischief, there were no symptoms which called for interference. The following day, however, the lad died suddenly with symptoms of collapse.

On examining the body, after death, there was found a general inflammation of the peritonæum, and the extremity of a loop of ileum was found caught and strangled in the left internal ring, and the bowel was perforated at the inner side of the protruding portion. As fully three-fourths of the wall was free from the stricture, there was a clear passage for the intestinal contents along the natural canal.

In both of these instances when the patient came under our observation, there was an entire absence of symptoms of hernia or of strangulated hernia, consequently the question of, or the necessity for, operating was not considered, but I would add that in the second, as in the case presently to be related, no operation would have increased the chances of recovery, and for this reason—either patient died from peritonitis radiating from the strangulated bowel, and spreading in every direction. There was no adhesion preventing its extension beyond the situation of the rupture, such as existed in the case first narrated, in which the woman's life was saved by the limit which Nature set upon the peritonæal mischief. Now, in such cases as these, when peritonitis spreads by continuity, it invariably proves fatal, and as the first symptoms which attracted serious attention to these cases were those of peritonitis, I consider that any cutting operation performed later would have only hastened the fatal result; it would have been absolutely fruitless in checking the peritonæal inflammation. Should a similar case come under the observation of any reader of these notes, he

may be content to remember that when no symptoms of a tumour or of strangulation mark the existence of a hernia, there is no ground for reproach that an operation has not been performed, even if it is shown by after-death examination, that a small portion of the bowel has been strangulated, and has occasioned the fatal peritonitis. In such cases part only, not the entire circumference of the wall of the gut, has been protruded, and as the fæces can pass through the canal at the back of the strangulated portion, neither vomiting nor constipation have been excited; should the peritonitis set up by the strangulation of the part of the circumference of the bowel be diffused by continuity, the disease is mortal; should it be limited, as in the first case, to gluing the intestine to the parietal peritonæum at the seat of the stricture, the patient will suffer from a fæcal fistula, but this will in all probability eventually close.

The third case allied to the preceding is the following. Dr. Andrew having asked me to see one of his patients, I went up to Mark Ward, and found a male, æt. 18, a messenger in the office of the 'Daily News,' and the following was given to me as his history. About a week previously, being then quite well, he was suddenly seized with severe griping pains in the lower and right part of his abdomen; these grew worse, and on the following day he began to vomit. Two days later the sickness, and with it the swelling of the abdomen subsided; later on he had some recurrence of sickness, although the pain in the stomach was less. It will be observed there was nothing in his history to mark the descent of a hernia, no strain, no mention of a tumour.

He lay on his back, being easy in that position only; his countenance showed freedom from pain and anxiety; his pulse was 90, and his tongue only slightly furred, but moist; the respiration was not hurried, but the breathing was thoracic. He was now free from sickness, indeed he vomited only once during the twenty-four hours after his admission, and the vomit was not stercoraceous. The abdomen was swollen but not very tense, and below a line drawn through the umbilicus into either lumbar region it was dull throughout, and everywhere tender to the touch. The bowels had not been properly opened for some days, but the evening before his admission he had passed a scanty motion. Micturition was frequent, but the urine passed was natural. I found that Dr. Andrew had already looked for a hernia, and on carefully examining the abdomen I agreed with his conclusion that no rupture existed,

and that there was no local pain to attract attention to any one part of the belly in particular. We discussed the possibility of there being an intussusception, and I examined the rectum on the question being raised that the prolapsed bowel might be detected there. As, however, no symptoms save those of peritonæal tenderness existed, we agreed to wait, giving the patient some opium, the dose to be repeated if necessary.

After this he slept well, and subsequently expressed himself as much easier than on admission, and there was no return of the sickness, but early in the morning he died quite suddenly. He had been ill nine days, of which he was for about thirty-six hours in the Hospital.

I am indebted to the kindness of Dr. Church for the following notes of the post-mortem examination.

Autopsy thirty hours after death. Body fairly nourished, dark green discoloration of the abdomen spreading upwards over the trunk. Rigor mortis considerable. Pleuræ natural. Lungs congested and œdematous posteriorly, their tissue not softened. Pericardium and heart natural. Liver, kidneys, and spleen all congested, but in other respects natural.

Within the cavity of the peritonæum a little dark-coloured serum was found. The serous coat of the intestines was extremely vascular and congested, and lines of greatly increased vascularity marked the spot where the coils of the small intestine came in contact with each other. The parietal layer of the peritonæum, in the pelvis and lower portion of the abdomen, was also vascular and congested. On examining carefully the small intestine a knuckle was found strangulated at the entrance of the right inguinal canal. The internal ring was open and led into a small sac, which passed down below the external ring into the front of the pubes. To the margins of this sac at the internal ring the gut was adherent, but not very firmly, pulling out with ease, and it was then apparent that only a portion of the gut was strangulated, at least one half of the circumference of the bowel not being embraced in the stricture. The strangulated portion was of a dark claret colour, but neither ulcerated nor sloughing; it was situated about four feet above the ileo-cæcal valve. The gut between the valve and the strangulated portion was empty and natural; above, it was distended by fluid fæces, and its mucous membrane ulcerated in patches. At one spot where the bowel was adherent to the descending colon by pretty firmly organised lymph, perfor-

ation had taken place, but there was no escape of fæcal matter into the cavity of the abdomen.

I hope it will be understood that I am quite conscious of the unfortunate nature of the history thus recorded; but from the attention bestowed upon the case by Dr. Andrew and by myself I am justified in the statement that no symptoms existed which would have warranted my cutting into the abdomen, for the signs of intestinal obstruction were far from clear, and of the existence of a rupture there was no evidence whatever. As before stated, this case may be grouped with the two which I now report as instances of masked hernia; they seem to me just the sort of records from which we learn to appreciate the difficulties which surround individual cases of strangulated intestine, and are illustrations of a class of cases which often pass under the observation of surgeons, but which from their unsuccessful results, are seldom placed on record for the instruction of others.

At one time or another I have seen a good many troublesome hernia operations. I will mention a few of these. The cases, I premise, are intended to illustrate the difficult questions which a surgeon may be at any time called upon to consider.

In the summer of 1863, I removed a scirrhous breast. The woman, æt. 42, went on well from the Saturday to the Friday following; she then, as she lay quietly in bed, suffered suddenly from all the symptoms of internal strangulation. No tumour protruded from the abdomen—nothing pointed to any one part of the abdomen as the seat of the mischief. Coming on as these symptoms did under such peculiar circumstances, they were much commented on, and there were some who suggested that the abdomen should be opened by an exploratory operation. However, I could not satisfy myself I should be justified in undertaking it, as there was no sort of guide, such as local hardness or pain, to direct my search. I think it was well left alone, for the post-mortem showed a round aperture in the middle of the mesentery, through which a loop of bowel had passed and become strangulated. It was deeply placed and covered by coils of small intestine, and could only have been found, if at all, during life after great disturbance and displacement of the parts.

I was recently led to perform an operation under the following circumstances—Mr. Square, the House Surgeon on duty, sent

for me on November 2, 1867, to see a female, æt. 37, who, whilst lifting a pail, suddenly felt something give way in the left groin. She at once detected a considerable swelling, felt local pain, and presently was sick and vomited. The following day, when she came to the Hospital, the symptoms persisted, and the bowels were confined. She did not look very ill, but in the face of this history, and finding a firm tumour (which had resisted taxis) in the inguinal region, I considered it right to determine its nature. In cutting down upon it, we found that it consisted of a clot of blood following the course of the round ligament, and firmly bound down by fascia. The next morning the patient began to menstruate, the first time since her last confinement.

Of so-called 'exploratory' operations, the following is an instructive example:—

Violent and continued sickness, with enlargement about the external abdominal and crural rings—Operation—Death from fever.

On September 10, 1856, a female, æt. 62, was transferred to St. Bartholomew's from the Fever Hospital, on account of sloughing of the integument of the right leg. She lay in the ward for three weeks, when suddenly she suffered from rigors, and was very sick. She passed a restless night, vomiting constantly. Pulse 100. Bowels not open for three days.

On examining the right groin, a small, soft, rounded tumour was felt in the situation of the external abdominal and crural rings. This received a distinct impulse, and was easily returned into the abdomen. There was felt below this a small, hard, body, in feel resembling a gland; this occupied the situation of the femoral ring. There was no tenderness in either of these situations, and the abdomen was everywhere soft and tolerant of pressure. She had never worn a truss.

October 8.—She remained in much the same state the whole of yesterday, the vomiting having been incessant. The matter ejected was of a yellow, bilious character; the bowels acted, however, freely in the course of the day. There was considerable tenderness at and around the femoral ring.

In the afternoon it was determined in consultation to explore the seat of the femoral tumour, as it was feared the extremity of a loop of intestine might have become strangulated there. The femoral ring was found occupied by an enlarged gland. The inguinal canal was free; a small sac contained neither omentum nor hernia.

9.—After a short interval the vomiting recommenced.

10.—An abundant rash of dark red elevated spots appeared on the surface of the abdomen and chest. The patient became delirious, and sinking rapidly with a continuance of the symptoms of fever, died on October 12.

A pad of omentum lying over a small knuckle of intestine will often prevent the reduction of the bowel by taxis. It is very vexing after having opened the sac, to see how slight has been the obstacle, but I do not know that it can be removed otherwise than by an operation, and the patients generally recover. During the last year I had to operate on a case of this kind. A female, æt. 42, had suffered from strangulation for two days and a half. The hernia felt as though it should be easily reducible, yet the sac had to be opened, and then a pad of omentum was found covering, and quite preventing pressure upon, a knuckle of sodden intestine, which on exposure was easily returned. The omentum sloughed out from the wound, but the patient made a good recovery. Such cases are of comparatively frequent occurrence. Mentioning the sloughing of the omentum reminds me of the necessity, as I consider it, of leaving a portion of the wound open, so that pus and other fluids may drain away, and not sink back into the peritonæum, or infiltrate the loose cellular tissue about the sac. I am sure I lost a patient by neglecting this simple precaution.

On February 13, 1864, a female, æt. 40, was admitted with a femoral hernia strangulated for three days. There had been no treatment before her admission, and after trying taxis, under chloroform, I operated, opening a thick walled sac, inflamed, and returning into the abdomen a small knuckle of intestine. The wound was then closed with sutures. She went on very well for a few days, but gradually symptoms of peritonitis supervened (not as from perforation of the bowel), and she died on the ninth day. There was general peritonæal inflammation, but the bowel had quite recovered itself, the incision into the sac had completely closed. I find the following added to the notes of this case: 'I cannot but think that unhealthy exudations within the sac, not being able to escape externally, trickled back into the peritonæum, and so aroused the serous inflammation.'

The sac in this case was inflamed and lined with lymph; in the following instance its inflammation, and that of some omentum within it, simulated a strangulated hernia. A female, æt. 58, was admitted during October 1851, with femoral hernia of three days' standing. There was constant vomiting, and

although the bowels had acted, only scanty scybala passed away. The tumour was very painful, and Mr. Stanley at once decided upon the necessity of operating, and having determined to open the sac proceeded, with his usual care, to remove layer after layer of cellular tissue; on a sudden we saw a quantity of whitish grumous fluid escape from the swelling, and we all thought that the intestine had given way. It proved, however, to be pus from the inflamed sac, and from some omentum, also inflamed, loosely contained within it. The omentum was cut away, and a clear opening felt which led into the cavity of the abdomen. This patient recovered slowly. In this case the omentum was doubtless rightly removed. Unless, however, it is unmistakably gangrenous, it should, as a rule, be left in the wound, and the wound should be left partly open for drainage. I never saw any result from removing omentum, except now and then a troublesome bleeding; I never saw any harm from leaving it in the wound; but I have seen the disastrous consequences of returning, even what one hears called 'not bad looking' omentum into the cavity of the abdomen.

The following case presents some unusual complications. In August 1853, a male, æt. 23, was admitted, under Mr. Paget, having suffered for two years from an oblique inguinal hernia on the right side. For two months he had had trouble in passing his urine, which trouble had materially increased during the last eight days, in consequence of a hurt to his perineum. For a like time his bowels had remained obstinately constipated, with vomiting and increasing abdominal pain.

In the perineum was found a soft, fluctuating tumour, which reached to and pressed in upon the rectum, where it was comparatively tense and hard. In the right groin, following the course of the cord, was an oval swelling, not tense, but extremely tender on pressure. It was just like a hydrocele formed around a testicle which had not completed its descent into the scrotum. However, either swelling had to be examined, as his symptoms had to be relieved. The tumour of the perineum was found to contain a large quantity of fluid blood; that in the groin was filled with turbid serum. On introducing the finger, the testicle was felt in the canal just below the spine of the pubes, separated by a fold of peritonæum from the sac just opened. Above, the finger was arrested by a small knuckle of intestine, strangulated at the internal ring. It was easily reduced, but the vomiting (stercoraceous) continued, and the patient died about thirty hours after the operations.

Post-mortem Examination.—There was general peritonitis, and parts of the intestinal canal were distended with flatus and fæces sufficient in places to cause compression of the folds of the gut; and to this was ascribed the persistence of the vomiting which had been so anxiously watched and considered during the last few hours of the patient's life.

No cases cause greater anxiety to the surgeon than do those in which symptoms of strangulation continue after he has operated, and has, as he believes, relieved the stricture of the gut. What occasions their continuance? will it be right to explore the cavity of the abdomen in the hope of finding the cause and of removing it? are problems which have occasionally to be solved. I do not, of course, deny but that such an exploratory operation is often justifiable, or that it has occasionally been successful, but the cases next to be related illustrate the extreme uncertainty which must always exist as to the cause of the persistence of symptoms of strangulation, and the unlikelihood of their being removable by surgical interference.

During March 1850, a male, æt. 45, was admitted with a large, tense, and exceedingly tender scrotal hernia of the right side, strangulated ten hours. The intestine was presently returned with the help of chloroform. The vomiting ceased, the bowels acted, and the patient felt comparatively well during the 16th, 17th, and 18th days of the month.

But on the 19th, no evacuation having occurred for two days, the pulse began to rise, not rapidly; he began to feel uneasy, and his abdomen grew somewhat tympanitic. He was given a purge (purgatives were employed for such cases in those days), and no great anxiety was felt respecting him; but on the 20th he vomited, and during the afternoon he was frequently sick, the matter rejected becoming stercoraceous. The abdomen became greatly distended, and the bowels were still confined. The pressure of symptoms became so great that Mr. Stanley considered it right to explore the hernial sac. It held some omentum but no intestine, and puriform fluid flowed through it from the cavity of the abdomen. All the symptoms continued unrelieved, and, sinking rapidly, he died the following evening.

The cause of the fatal result was anxiously looked for at the post-mortem examination. There was general peritonitis, and one mass of ileum (about the size of the closed fist) was rolled up, and bound by peritonæal adhesions, so that no fæces could

pass through its canal, but its wall was nowhere strangulated. The adhesions, it was noticed, were so firm that the walls of the intestine tore before the lymph gave way. Had the abdomen been opened more freely, and this mass of ileum exposed, it would have been impossible to have relieved the obstruction.

The following cases illustrate a form of strangulation not very frequently observed, and which has not, that I am aware of, been described in connection with the occurrence of hernia.

About 2 A. M. December 11, 1866, I operated at St. Bartholomew's on a male æt. 22. He had, so he told us, been ruptured two years, and had worn a truss. The hernia came down about sixty hours ago and could not be returned. It was a large scrotal hernia, congenital. Mr. Humphry, the house surgeon, having tried the taxis, I proceeded to expose and divide the external ring; this enabled us to detect a dense constriction in the sac, which could only be divided by cutting through the peritonæum. The bowel, not bad looking, was then easily returned; the omentum was left in the wound, being adherent below to the sac just above the testis. The wound was only partially closed to allow of drainage.

The bowels acted scantily on the third day, but the sickness and vomiting persisted, and he sank and died four days after the operation. We found that there was general peritonitis. A cord-like band of omentum extended from the stomach to and along the canal, adhering to the hernial sac. It was inflamed and suppurating. But round this the bowel, ileum, had taken a turn and a half and thus twisted had suffered complete occlusion of its canal. If I had opened the abdomen and exposed this portion of its contents, I could not have relieved the bowel, so entangled was it, and so matted to the adjoining structures.

I kept a note of a very similar case which was under the care of Sir William Lawrence. A female, æt. 34, was operated on for a femoral hernia of the right side, strangulated for eighty hours. The sac was at once opened. It contained about half an ounce of turbid serous fluid. A piece of thick indurated omentum was exposed, fastened to the anterior part of the ring by old adhesions. There was no intestine in the sac, and the finger could be passed easily into the cavity of the abdomen, but nothing could be felt of any strangled bowel. Two hours after the operation she was sick twice and vomited fæcal matter. The bad symptoms of strangulation persisted until she died the following afternoon.

At the post-mortem examination it was clear that no operation could have afforded relief. The peritonæum was univer-

sally inflamed. The inflammation was most intense in the right iliac region. The greater part of the omentum extended as a thickened band from the stomach to the femoral ring, dragging the stomach downwards. A large quantity of small intestine lay in front of the omentum, and a coil of this portion had slipped so far round as to get beneath the omentum again, and was there strangulated between its own mesentery and the omentum, being also twisted upon itself. The strangulated portion, about three inches long, was a piece of the ileum two feet from the cæcum. The strictures were tight, and beneath them the bowel was ash-coloured. No intestine was protruded from the abdomen.

This was in February 1854, and the notes were given to me by Mr. F. A. Humphry, who was then house surgeon.

ARTICLE XVII.—*Notes suggested by some of the Specimens that have been added to the Museum in the course of the year.* By F. H. MARSH.

THE term 'loose cartilage' is convenient, because everyone understands that it is applied collectively to those bodies which are occasionally found, now singly, now in considerable numbers, in the cavities of joints. But with regard to the origin and composition of these substances, the term is often inaccurate; and it is so generally recognized that this is the case, that many writers who use it place it between inverted commas, as if it were accepted under protest, or at least with a reservation. The Germans have named these bodies 'joint-mice' (Gelenkmäuse) from the fact that when any attempt is made to secure them, they immediately retreat to some recess in the articular cavity where they cannot easily be found. Several distinct varieties have been described; but as the formation and composition of others are still under discussion, and as the subject was particularly suggested by a remarkable specimen removed by Mr. Holden from a man's knee-joint a few months ago, I have examined all the examples that are contained in the Museum, and shall, a little further on, offer a brief account of their structure.

I. Hunter * believed that loose cartilages are originally small masses of coagulated blood, shed into the joint in consequence

* Works of Hunter, edited by Palmer, vol. i. p. 520.

of injury. He says—'This (blood), I suppose, when it is extravasated into a cavity, adheres to the cavity, and is not absorbed, nor loses its living principle, but becomes vascular, and afterwards membranous, cartilaginous, or bony, according to circumstances. Its attachment being rendered small by motion, it becomes easily broken off.' And Sir E. Home* gives the following account of the dissection of a false joint formed at the seat of an ununited fracture of the humerus, which he considers 'exhibits so many facts in confirmation of this (Hunter's) theory, that it appears to afford a full explanation of the process above mentioned, and completely to establish the opinion.' 'The surfaces of the bones fitted for motion were not completely covered with cartilage, but studded over with it, and the bone was exposed in the interstices; a number of projecting points covered with cartilage grew out from the surfaces, some exceedingly small, others large. From the edges of the bones and the capsular ligament these excrescences were larger, extremely irregular in their shape, broader in their attachments, softer in their texture, and serrated upon the external edge. Thirty or forty small substances, similar to those above mentioned, were found loose in the cavity, varying in size from that of a millet seed to that of a barley corn, of a roundish form and smooth on the surface; the largest of them were much flattened and serrated. Their hardness varied considerably, some of them being as soft as cartilage, others so solid as not to be pierced by a needle.' And he continues: 'Those bodies must have been originally attached, and broken off by the friction of the parts on one another. After the time of the injury some of the extravasated blood was absorbed, while the remainder underwent changes in its nature, assimilating it as far as possible to that of the surfaces to which it was attached, in some its tissue resembling ligament, in others being more allied to cartilage.' No doubt extravasated blood sometimes, instead of being, as it commonly is, absorbed, undergoes development into fibrous tissue; † but whether pathologists of the present day believe, with Hunter, that it may become cartilage or bone, and so form these loose bodies in the joints, I dare not say. But, to say the least, this is not the manner in which these substances are commonly produced. And it may be well to separate Hunter's opinion from Home's supposed demonstration of its accuracy. I say 'supposed demonstration,' because Home begs the whole question by assuming

* Loc. cit. vol. iii. p. 628.

† Paget, 'Surgical Pathology,' 1863, p. 131.

what he should have proved, that the nodules of cartilage which encrusted the ends of the fragments, in the case which he describes, had been developed from extravasated blood. There is nothing in his statement to forbid the belief that would accord with Mr. Paget's observations * on the repair of fractured bones and severed tendons, that any blood which was shed between the fragments at the time of the injury, and which was probably not large in amount, was absorbed, and that the cartilage found on the ends of the fragments was produced by the development of a special reparative material. In assuming that this cartilage had been produced from extravasated blood, Home leaves the exudation of a special reparative material wholly out of account. And is not his reasoning a little in advance of his facts, when he says that 'the remains of the coagulated blood underwent changes in its nature assimilating it as much as possible to that of the surfaces to which it was attached, in some parts its texture resembling ligament, in others being more allied to cartilage or bone'—for there was, of course, no cartilage on the ends of the fragments to which the blood adhered?

But to return to Hunter. I am not aware that it has ever been proved that those loose bodies in joints which have a true cartilaginous structure are ever developed from extravasated blood. Indeed the difficulty of proving this occurrence must always be great, for an injury that would produce extravasation of blood into a joint would, in all likelihood, be followed by some inflammatory action; and as the history of many of these cases† shows that 'loose cartilages' usually arise as the consequence of inflammation after injury, it would be hard to demonstrate that these bodies are produced from the extravasated blood, and not by an overgrowth of the synovial fringes (see p. 251).

II. Many writers agree in the belief that, in some cases, these bodies originate in masses of fibrin which have been effused during inflammation. Thus, Gross‡ says, 'without recounting any of the speculative views that have been advanced upon the subject . . . it must be sufficiently apparent to every pathologist that these bodies invariably take their rise in plastic effusions excited under the influence of inflammation.' Rokitansky§ states that 'some of them are masses of exudation set

* Loc. cit. pp. 133–136.

† Hey, 'Pract. Observ. in Surgery,' 1810, pp. 343–351; Abernethy, Surgical Works, 1819, vol. ii. p. 215; Square, 'London Med. Review,' Oct. 1861, p. 167.

‡ 'Syst. Surgery,' vol. i. p. 998.

§ Path. Anat., 'Syd. Soc. Trans.' vol. iii. p. 295.

free;' and Kölliker* is 'inclined, with Virchow, who has actually demonstrated the presence of fibrin in them, to regard them in many cases as fibrinous exudations.'

III. Both Rokitansky† and Kölliker‡ think that in other instances they consist of mere precipitates from a depraved synovial secretion, and such, Rokitansky says, are sometimes laminated. I have not myself had an opportunity of examining either of these varieties, but they are no doubt very similar, both in origin and composition, to those bodies like melon-seeds which are contained in bursæ and in the sheaths of tendons; such, for example, as I found in a large and very thick-walled bursa patellæ, removed during the present year by Mr. Holden.§ It contained a clear yellowish-brown viscid fluid, in which floated a number of pinkish, semi-transparent, glistening bodies of various shapes and sizes, and presenting a nodulated, mulberry-like surface. Many of these bodies floated in the fluid, some isolated, many loosely fastened together by fine fragile membranous pedicles; others were in the same manner attached to the internal wall of the bursa. All the pedicles were very easily broken, so that the bodies became completely detached. Under the microscope these substances were found, for the most part, to possess a granular, amorphous structure, but here and there an indistinct fibrillation was observed; no trace of cells, or of fibres that could be isolated, was seen.

Perhaps the following specimens, in the Museum of St. George's Hospital, which are described as consisting of fibrous tissue, may have originated in fibrinous exudations.‖ 'Two loose cartilages from the knee. They consist of finely fibrillated nuclear blastema, without cartilage cells. The central part of each is calcified.'¶ A loose cartilage from the knee. It consists essentially of fibrous tissue, without any amorphous granular blastema or cartilage cells. Its centre is calcified.'

IV. There can be no doubt, in spite of the somewhat confident statement of Gross, mentioned above, that some, indeed the large majority, of the so-called 'loose cartilages' are produced by an abnormal growth of fine processes of the synovial membrane. Rainey** describes on the inner aspect of the synovial membrane 'loops, and convolutions of capillaries .... projecting into the synovial cavity, and enclosed in sacculi of basement membrane, studded with minute oval

* Human Histology, 'Syd. Soc. Trans.' vol. i. p. 329.
† Loc. cit. vol. iii. p. 41. ‡ Loc. cit. § Series V. 38.
‖ Series III. 138. ¶ Ibid. 139. ** 'Path. Trans.' vol. ii. p. 110.

cellules. From the sacculi enclosing capillaries numerous other sacculi proceed; these are of various forms and sizes, but are generally attached to the primitive sacculus by an extremely long and slender filament of fibrous tissue resembling the petiole of a leaf, the secondary sacculus being its expansion. Sometimes there are several series of these sacculi, attached one series to another, exhibiting an arborescent appearance, but in every instance the secondary sacculi are extra-vascular. . . . . Now, this being the apparatus by which synovia is elaborated . . . . the cellules of these fringes, in place of elaborating synovia from the blood, produce under the influence of morbid action other products, such as cartilage, which becomes converted into imperfectly-formed bone. The fact of the secondary sacculi being connected to the primary by extremely narrow pedicles will suffice to explain why these bodies may become formed in the first instance, the pedicles serving both to keep them attached, and to convey the material from the blood necessary for their development until they acquire a certain size; but afterwards from its tenuity, being no longer capable of holding, it breaks, and the bodies become loose, and most likely cease to enlarge.' Kölliker* says: 'The synovial membranes possess neither glands nor papillæ, whilst they present large adipose masses (plicæ adiposæ) and vascular processes (plicæ vasculosæ). The former . . . consist simply of large collections of fat-cells in vascular portions of the synovial membrane. The latter . . . constitute, provided the vessels are filled, red flattened projections of the synovial membrane with an indented plicated margin, and furnished with minute processes. . . . In their structure they differ from the rest of the synovial membrane principally in their great vascularity, consisting as they do of little else than minute arteries, and veins, and delicate capillaries, forming wavy loops at the edge of the processes, and consequently they are very similar to the choroid plexuses of the ventricles of the brain. Besides the vessels, they present a matrix of, frequently distinctly fibrous, connective tissue, the usual epithelium of the synovial membrane, and more rarely isolated cartilage cells. At the edge they are almost invariably furnished with minute foliated, conical, membranous processes of the most extraordinary forms (often resembling the stem of a cactus), which also frequently contain vessels, but for the most part are constituted merely of an axis of indistinctly fibrous connective tissue, with occasional cartilage cells, and

* Loc. cit. vol. i. p. 326, et seq.

an epithelium very thick in places.' And he states* that the non-vascular appendages of the processes of the synovial membrane give origin to the 'loose cartilages,' as they are termed; they do this by increasing in size and solidity, and becoming detached from the vascular folds.

The formation of these bodies, in the manner described by Rainey and Kölliker, is admirably illustrated by two specimens in the Museum. The first† is a shoulder-joint laid open, from a patient who had suffered from chronic rheumatic arthritis. The inner surface of the synovial membrane is thickly set with numerous club-shaped, or villus-like processes. Many of these I found with a microscope to consist apparently of fat and fine blood-vessels, enclosed in a fine basement membrane. Others, the largest of them, were composed of perfectly formed fibro-cartilage. The second‡ is an elbow-joint laid open. Attached to the margin of the greater sigmoid cavity of the ulna by a pedicle of the thickness of fine string, and about one-third of an inch long, is a pear-shaped nodule of fibro-cartilage of the size of a small bean. The pedicle is so slender that it is evident how easily the body might have become entirely loose in the joint.

Rokitansky§ agrees with Laennec that in some cases these bodies are originally developed in the subsynovial tissue, and, gradually forcing the membrane before them, at length project into the cavity of the joint. And Sir B. Brodie,‖ after stating that they generally originate in the synovial membrane and then break off, goes on to say that 'some in St. George's Museum¶ are seen to be developed outside the synovial membrane in contact with the subsynovial tissue; some of these are partly bone and partly cartilage, others are wholly cartilaginous.' While, in opposition to this view, Kölliker says: 'They are not developed externally to the synovial membrane, but from an outgrowth from that membrane itself.'** But that these bodies may originate in the subsynovial tissue and then project into the joint is highly probable, for in many cases of chronic rheumatic arthritis plates and nodules of fibro-cartilage are found in this situation, and some of them may be seen projecting towards the joint cavity.

There are no means of knowing with certainty how the following five specimens were formed, but as they appear to belong to the variety just described—to those, namely, which

* Loc. cit. p. 328.
† Series II. 22.
‡ Ibid. 39.
§ Loc. cit. p. 41.
‖ Surgical Works, vol ii. p. 284.
¶ Series III. 143.
** Loc. cit. vol. i. p. 329.

are produced in the synovial membrane or in the subsynovial tissue—their microscopic structure may be here given.

1.*—A thin flattened oval body, measuring about an inch in its longest diameter. It was removed from the knee-joint. It consists of well-formed hyaline cartilage. The matrix is coarsely granular, but presents no trace of fibrillation. The cells are precisely similar to those of ordinary hyaline cartilage, and lie thinly scattered, either singly or in groups of two or three. The central part of the cartilage is calcified, and very hard. Treated with dilute hydrochloric acid, the structure is seen to resolve itself into pure cartilage, exactly similar to that found in the uncalcified portion, both the cells and the matrix becoming perfectly distinct. Many of the cells in the centre are large, and crowded with small round or oval nuclei.

2.†—A similar, but rather smaller specimen. The matrix is hyaline. The cells are, in their general form, like those of hyaline cartilage; but they vary much in their size and shape in different parts. In some districts they are large and very elongated, and filled with nuclei, while in others they are small, thickly placed, and like those of fœtal cartilage. Only the central portion is completely calcified, but specks of earthy deposit are scattered through its whole substance.

3.‡—This specimen consists of fifteen loose bodies that were removed from a hip-joint. No history of the case is known. They are all alike in structure, and vary in size from a small nut to a chestnut. They are formed of irregular masses of hyaline cartilage, variously mixed with fibro-cartilage. On section they are found to have an open broken structure from the presence of numerous holes and interspaces, like masses of dry earth, and they are friable, and easily 'crumble' down under the finger. Microscopically the matrix looks confused and degenerate, and the cells are ragged and broken and indistinct. To the touch these bodies are quite stony, and rattle like stones when dropped upon earthenware.

4.§—A flattened, nearly circular, concavo-convex body, an inch and a quarter in its greatest diameter, removed from the knee of a lad, æt. 17, in whom the symptoms had existed for upwards of two years. The matrix is found to be for the most part hyaline; but near the surface it is distinctly striated, so as to resemble that of fibro-cartilage. The cells (or nuclei) are small, oval, and so closely packed that in some places very

* Series II. 25. † Ibid. 26. ‡ Ibid. 66. § Ibid. 74.

little matrix can be seen between them. In many parts small specks and patches of calcification are observed; but when the section is treated with dilute hydrochloric acid, the original cartilaginous structure becomes perfectly distinct.

5.*—Is of the size of a short thick almond, and of an irregular outline. It was removed from the knee-joint of a boy, æt. 15, in whom the symptoms had existed five years. It has a fibro-cartilaginous structure. Towards its surface it is distinctly fibrillated; well-defined fibres, and groups of fibres run variously interlacing with each other. Among these fibres lie thinly scattered nuclei, or cells, oval or oat-shaped. In its interior the cartilage has a hyaline matrix, in which lie groups of oval cells containing numerous well-defined and variously-shaped nuclei. This cartilage has a cavity in its interior formed by the 'blistering up' of a thin layer from the rest of the substance. This raised portion consists of fibro-cartilage.

V. Another form of 'loose cartilage' consists of masses that are derived from what Dr. Robert Adams† has termed additamentary bones, which are formed around the margins of joint-cavities, or upon the articular extremities of bones, in the course of chronic rheumatic arthritis. These growths are occasionally broken from their attachments, or pieces are broken off from them, and in either case the masses become loose in the joint. Such bodies are irregular in size, some being as large as, or larger than, a walnut, while others are very small. Their surface is very irregular. Many of them are harder and denser than normal bone; some may be easily cut with a knife, and are found to have a granular powdery structure, like dried mortar; and they may occur singly or in considerable numbers.

VI. The variety of 'loose cartilage,' to which I wish to draw especial attention here, is one that has been referred to by several writers,‡ although its occurrence has never, I think, been generally acknowledged. It is that which results from the breaking-off, or shedding into the cavity of a joint, of a portion of the proper articular cartilage, together with a layer, more or less substantial, of the subjacent bone. Brodie says: 'In one case in which I had an opportunity of examining the parts, by dissection, besides some loose cartilages having the usual appearances, I found another loose body of an irregular

* Series II. 77.

† 'On Rheumatic Gout,' p. 33.

‡ Brodie, loc. cit.; Reimarus, 'De Fungo Articulorum'; Square, loc. cit.; Virchow.

shape, with one surface smooth and cartilaginous, and the other surface having a thin layer of bone adhering to it, being evidently a portion of the articular surface actually broken off from the head of the tibia. That such an accident should occasionally happen from any sudden or violent motion of the joint, with a hard body loose in its cavity, is no more than might reasonably be expected.' The presence, as in this instance, of a loose body in the joint would doubtless make the accident more likely, but that it may occur in a previously healthy joint I think is shown by the cases that follow.

A bargeman, æt. 27, was admitted into one of Mr. Holden's wards, in May 1868. He had been suffering severely from the ordinary symptoms of loose cartilage in the left knee for about three months, and during this time he had often felt a foreign

Fig. 1.

Fig. 2.

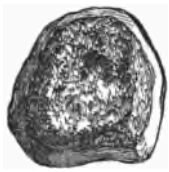


Fig. 3.

Fig. 4.

body freely movable in the joint. He was not aware of having injured his limb. As he was disabled by frequent attacks of pain and lameness, Mr. Holden removed the body (which was readily found) by Goyraud's subcutaneous operation. The patient had no bad symptom, and was soon fit to leave the Hospital. The size and appearance of the body are shown in figs. 1 and 2. It consists of a layer of cartilage and a layer of bone intimately connected with each other. The cartilage is smooth and glistening on its free surface, like ordinary articular cartilage; in profile it is seen to have the thickness of the layer that encrusts the condyle of the femur in a healthy adult; its margins are irregular, as if defined by fracture, and receding towards the bony layer. This latter is of less superficial extent than the cartilage, which exceeds it in

every direction. At its central, which is also its thickest, part, it measures about two lines; its surface is irregular and cancellous (fig. 2). Examined with a microscope, the cartilage is found to be precisely like articular cartilage. In its deeper layer the cells lie with their long axis at right angles to the subjacent bone; while, towards the free surface, they are small, numerous, elongated, and with their long axis parallel with the surface. The bone presents the characters of true osseous tissue.

Two portions of cartilage now in the Museum* were removed some years since by Mr. Holden from the knee-joints of a lad, æt. 18. 'They are almost exactly alike in form and size, each resembling such a piece of cartilage as might be obtained by removing that which covers the posterior surface of one of the condyles of a femur; and each, as such a piece would be, is smooth and polished on its convex, and rough on its concave, surface. There was an interval of about a year between the operations by which these bodies were removed from the joints. The patient recovered from both operations without an untoward symptom.' On close examination both these cartilages are found to have a thin layer of bone on their concave surface (fig. 4); this presents, under a microscope, the characters of true bone. It is, however, somewhat softer than healthy bone, and is covered with a scanty layer of irregularly disposed fibrous tissue. The arrangement of the cells in the cartilage of both these specimens is precisely similar to that of articular or encrusting cartilage.

In the Museum of St. George's Hospital† is the condyle of a femur with two loose cartilages. 'A large cavity is seen in the cartilage covering the condyle, about an inch in length and three-quarters of an inch in breadth. The articular lamella is exposed at the bottom of the cavity in the greater part of its extent. The cartilage surrounding the margin of the cavity presents a puckered and cicatrized appearance, except at one point where it is partially separated from the articular lamella beneath. Of the two loose cartilages, one is an irregular piece of cartilage; one surface is marked by elevations and indentations, corresponding to the articular lamella. Part of the margin of the loose piece is rounded as if by absorption. The remainder of the margin presents a broad uneven edge. . . . The second piece of cartilage is comparatively smooth, and consists of cartilage partially calcified. The patient was a young woman, about twenty years of age.'

* Series XXXV. 55. † Series III. 140.

In attempting to establish an alleged fact, the usual course is to throw out a theory that may make it look probable. In the present instance I can only give an account of an examination of these specimens, and there leave the matter. Under what circumstances portions of cartilage and bone are broken off, or otherwise separated from the proper surface of a joint, I cannot tell. It is difficult to see how such a mass as is shown in figs. 1 and 2 could be detached, and also how the joint from which it was derived could, after such an injury as the composition and size of the body imply, escape extensive inflammatory disease.

A specimen * in the Museum of the College of Surgeons is in itself remarkable, but I copy its description from the Catalogue, because it serves to illustrate how strange an injury a joint may sometimes tolerate.

'A knee-joint in which there are two small growths of apparently cartilaginous and osseous substance. The largest of them, attached to the crucial ligaments, has an oval outline, measures nearly an inch in its greatest diameter, is flat on its lower and very convex on its upper surface. By friction and constant pressure it had worn for itself a deep cavity, with hard grooved walls, in the posterior and lower part of the outer condyle of the femur, in which it lay embedded and apparently immovable. The second, which presents nodules of cartilage on its surface, is fixed to the synovial membrane just below the patella.' The encrusting cartilage in this joint is much worn away, and the surface left by its removal is eburnated; but there does not appear to have been any inflammatory action as the result of the constant irritation which the pendulous cartilage must have produced.

Chronic rheumatic arthritis of the shoulder-joint—Separation of the acromion process. †

The scapula of a man who was brought to the room for dissection, and who had evidently suffered long and severely from chronic rheumatic arthritis. Unfortunately nothing was known, or could be learnt of his history, and the body had undergone such rapid putrefaction, after death from phlegmonous erysipelas, that no systematic dissection could be made, and only the left scapula was examined. The chief point of interest in the specimen lies in the condition of the acromion process. Dr. Robert Adams, of Dublin, says, in his work on Rheumatic

* No. 995. † Series III. 142.

Gout:* 'In cases in which the shoulder has been long the seat of this disease (chronic rheumatic arthritis) the acromion process has been found traversed in the original line of junction of its epiphysis by a complete interruption of its continuity, as if fractured. I say as if fractured, for I am convinced that this solution of continuity of the acromion process is not really a fracture produced by violence, but a lesion, which so frequently exists in combination with chronic rheumatic arthritis of the shoulder that I feel compelled to look upon it, in these cases, as a peculiar organic change, the result of chronic rheumatic disease. I do not pretend to account for the separation of the acromion process into two portions, nor can I see why it is that the division occurs in the original line of junction of the epiphysis, particularly at the late period of life at which we generally notice this phenomenon. Professor Smith agrees in the view that the separation in the cases in question almost invariably occurs where in early life the epiphysis joins the remainder of the acromion process, but still mentions that he has 'in one instance found the entire of the acromion separated from the spine of the scapula.'† In the specimen above referred to, ‡ at one inch from its extremity the acromion is completely divided transversely, but in the recent state the fragment was maintained in its proper position by means of ligamentous tissue, so that no displacement had occurred. At a distance of three-quarters of an inch further on towards the spine the acromion is again almost completely divided in a transverse direction, the two portions being held together by two narrow bridges of bone at their anterior and posterior margins. Much new bone has been formed about the glenoid cavity (which is much enlarged and greatly altered in shape), and the long head of the triceps muscle is extensively ossified. It is doubtful whether the second division of the acromion has ever been complete, or whether a partial reunion by new bone has followed a once completed separation. But this uncertainty does not seem very material. Whatever may be the explanation of the fact, as pointed out by Dr. Adams, that the separation of the acromion in these remarkable cases usually occurs in the position of the epiphysial line, the present case agrees with that of Dr. Smith in showing that the separation may take place at some other part of the bone, and perhaps the two cases together may be regarded as throwing

* P. 107.

† Smith On Chronic Rheumatic Arthritis of the Shoulder, 'Dublin Quarterly Journal,' May 1853. ‡ Series II. 142.

some doubt on the proposition that there is any physiological connection between this separation of the acromion and its former condition as an epiphysis.

*Abscess in the hip-joint—Separation of the head of the femur at the epiphysial line.**

The head of a femur, which became separated from the neck of the bone at the epiphysial line in the course of an attack of acute pyæmia. It was found lying loose in the cavity of an abscess which formed in the hip-joint, and it was removed at the time the abscess was opened. The patient, a boy aged 17, who was under the care of Mr. S. Rhind, recovered.

A somewhat similar specimen,† which consists of the head and part of the neck of a femur. The head has become separated from the neck at the epiphysial line; the neck is extensively ulcerated on its surface. The parts were removed from a limb which had been amputated at the hip-joint, on account of acute ostitis of the femur, by Mr. Rhind. The patient, a lad of 14, made a rapid recovery.

Malignant disease of the sigmoid flexure—Rupture of the large intestine from extreme distension.‡

Part of the sigmoid flexure of the colon, laid open. A soft, villous, malignant growth, springing from the mucous membrane, surrounds and almost completely obliterates the canal. The specimen was removed from the body of a woman aged 49, who having had for nine weeks occasional abdominal pain, and constipation which at length amounted to complete obstruction, was suddenly attacked with extreme pain in the abdomen, and faintness, and died in a state of collapse in about three hours. On dissection it was found that the transverse colon had become drawn down to the pubes in the form of a V, by a short piece of omentum that was adherent within the sac of an old femoral hernia. The colon thus displaced, had, on the occurrence of obstruction at the sigmoid flexure, become enormously distended and then inflamed, and at length the softened coats had given way by an extensive thin-edged ulcer or slough, through which large quantities of fæces had passed into the cavity of the peritonæum. The whole of the displaced colon had the appearance of having

* Series II. 81. † Ibid. 82. ‡ Series XVI. 135.

become completely paralysed by distension, as the urinary bladder may, and then to have passed into a state of low inflammatory softening, so that wherever it was handled it gave way. The patient was not seen till the alarming symptoms of extreme pain and faintness had set in; but had this been otherwise, it is probable that it would, on account of the state of the large intestine, have been useless to open the bowel above the point of obstruction. It is important to notice that this patient had continued to do household work up to within a few hours of her death. She had not believed herself seriously ill; for there were no symptoms to warn her, except occasional pain, and troublesome constipation. In her general health she had considered herself about as well as usual.

*Strangulated femoral hernia—Perforation of the intestine by a piece of bone that lay in its canal in the hernial sac.**

A portion of small intestine. Near its free margin, opposite the attachment of the mesentery, and about an inch apart, are two small ragged openings, through which a bristle is passed. The specimen was removed from the body of a middle aged man, who died under the following circumstances: A femoral hernia, which though of long standing had always been easily reducible, became strangulated. Its reduction was attempted unsuccessfully both by the patient and by a surgeon whom he consulted. He was then brought without loss of time to the Hospital. The hernia was of about the size of an egg; the skin over it appeared unaltered. The patient's general condition seemed good. He was placed in a warm bath, and a further attempt was made at reduction with the taxis, but this also failed. During the trial the patient complained of very little pain, and there seemed nothing that marked the case as one of unusual peril, or as in any way peculiar. Mr. Holden then proceeded to expose the hernia by opening its sac. The intestine, which looked only congested, was quickly returned. When this had been done, a substance, apparently a piece of bone, was discovered behind, and just external to, the hernial sac. This on being removed was found to be part of the rib of a rabbit, about an inch and three-quarters long, with sharp fractured ends. The patient died of peritonitis. On dissection a portion of small intestine was seen to be intensely inflamed, and on close examination the two

* Series XVI. 136.

openings above described were discovered; and there was also a perforation of the posterior wall of the hernial sac. It is supposed that the man swallowed the piece of bone in his food, and that this passing along the intestinal canal had lodged in that portion which formed the hernia. In the attempts that were made at reduction with the taxis the bone had been driven through the intestinal coats, and through the posterior wall of the sac into the cellular tissue just behind it.

*Keloids in the lobules of the ears following the use of earrings.**

Two fibrous tumours, of the size of large marbles, and nearly as round, which grew in the lobules of the ears of a young woman who had worn earrings. They are smooth on the surface—to which the skin is slightly adherent—firm and tough in their texture, and before their removal they looked dusky pink, like masses of keloid. The ears had been pierced two years before, and had remained in a state of chronic inflammation around the punctures for some time subsequently, and in the course of this the tumours were first observed. They were removed by Mr. Paget. A similiar specimen to this † was some years ago presented to the Museum by Mr. Holberton. It is formed of 'the lobules of the ears of a young woman. Within each lobule there is a nearly spherical fibrous tumour, over which the integument is tightly stretched. A section of one of the tumours displays a dense fibrous texture. Above these (in the bottle) is a small tumour of the same kind, which was reproduced in the cicatrix formed after the excision of one of the lobules. The tumours began to grow shortly after the patient began to wear earrings. The tumour last mentioned was removed shortly after its first appearance.' ‡

Congenital hypertrophy of the toes.§

A young sailor, aged 22, was admitted into Darker Ward, under the care of Mr. Paget, on account of a deformity of his left foot. The three intervening toes and the corresponding portion of the metatarsus were of an enormous size, and the like portion of the ball of the foot was covered with a huge cushion of fat. There was no appearance of disease in these toes, which seemed to owe their great size to what Dr. Car-

* Series XXXV. 135. † Ibid. 24.

‡ For a full notice of these tumours see Paget's 'Surgical Pathology,' 1863, p. 485. § Catalogue II. Series V. 219.

penter * has termed a 'preternatural formative capacity,' which was congenital in them, and by virtue of which they achieved a growth out of proportion to the natural dimensions of the rest of the limb. The patient found the deformity such an encumbrance, that he accepted Mr. Paget's advice and submitted to amputation of the overgrown parts. He recovered.

Fatal hæmorrhage from a blood-cyst in the broad ligament. †

The uterus and its appendages. The uterus is of the natural size in the unimpregnated state. The ovaries are very small, tough, and indurated, and their surface is deeply and irregularly scarred. On section, in the recent state, both contained shrunken pale corpora lutea; but no evidence was found of the recent bursting of a Graafian vesicle in either. Between the layers of the right broad ligament, just below the outer third of the Fallopian tube, is a globular cyst, about as big as a walnut, whose walls are thin and apparently formed mainly by the separated layers of the ligament, and whose cavity was found filled with pretty firm, and quite recent blood-coagula, of a dark colour. A careful microscopic examination of the coagula failed to detect any organised structure. On the anterior aspect of the cyst are two small, irregular, ragged openings. The patient from whom the specimen was removed was 25 years old, and was admitted into the venereal ward for the treatment of warts upon the vulva and perineum. She was a robust and well-nourished person, apparently in very good general health. The warts were removed under the direction of Mr. Callender, and after a fortnight's stay in the Hospital she was about to be made an out-patient, when one day, while at the closet at the end of the ward, she became suddenly faint, and complained of sudden and severe pain in the lower part of the abdomen. When she had been brought back to bed, it was seen that she was still very pale and faint, and Mr. Butlin, the House Surgeon, saw her, and believed that she was suffering from some form of internal hæmorrhage, but no blood was discharged externally. She was carefully treated with all the usual means, but the symptoms of internal hæmorrhage grew hourly more certain and more formidable, and at the end of twelve hours she died. I made an examination twelve hours after death. The surface of the body was extremely blanched. The viscera of the thorax were perfectly healthy. The peritonæal

* 'Human Physiology,' 1864, p. 350. † Series XXXI. 36.

cavity contained about five pints of blood very loosely coagulated, which lay chiefly in the pelvis; and dark coloured liquid blood was found to ooze slowly, on pressure, from the two ulcerated openings in the cyst of the broad ligament, already described. The cavity of the uterus, as well as all the other parts of the body, was very pale; but a small blood-clot lay along the canal of the cervix uteri. No blood was found in the vagina.

After a very careful examination of the blood-cyst I failed to find the source of hæmorrhage. I could not detect any evidence that the case was one of extra-uterine pregnancy, or trace any vessel that had given way. All the viscera were so blanched that all evidence of a congestion that might have attended a recent menstruation was destroyed. No doubt the case was one of those termed 'peri-uterine hæmatocele,' and it may serve to illustrate two points: 1. The extremely perilous nature of some of these cases of accidental hæmorrhage in the neighbourhood of the uterus; 2. The point insisted on by Dr. Tuckwell * and other authors, that in the large majority of instances the blood is poured into the cavity of the peritoneum, not into the sub-peritoneal tissue.

A few months ago I dissected another specimen, sent to the Museum by Mr. John Havers, from a case of peri-uterine hæmatocele. The patient had suffered for some months with repeatedly recurring attacks of moderately severe peritonitis. An attack more severe than usual produced her death; but, just before, it was observed that she passed urine loaded with broken-down blood clot. All the pelvic viscera and the small intestines were matted together by adhesions, some of which were old, others recent. A large cavity with irregular walls was found between the uterus and upper part of the vagina, and the bladder; and between this and the bladder was a small ulcerated opening, which had allowed its contents, formed of blood-clot débris, to escape into the cavity of the bladder. No extravasation of urine had apparently occurred. In this instance the blood occupied the more unusual position—that is, it was extra-peritonæal.

A calculus from a female bladder.†

It is somewhat pear-shaped and flattened, slightly rough on its surface, and composed of friable phosphates deposited upon

* 'On Effusions of Blood in the Neighbourhood of the Uterus, or the so-called Peri-uterine Hæmatocele: a Thesis for the M.D. Degree.' By Henry M. Tuckwell, M.A. M.B. Oxon. 1863.

† Series XXXVI. 212.

a small nucleus of oxalate of lime. It measures one inch and three-fifths in length, one inch and one-fifth in width, and four inches in its greatest transverse circumference. The specimen derives its interest from the fact that it was extracted through the urethra, which had been rapidly dilated with the finger, the patient quickly recovering, and subsequently retaining, perfect control over the passage of urine from the bladder. Presented by Mr. S. Rhind.

Article XVIII.—*On Rickets. Further Observations.*

By Samuel Gee, M.D. Lond.

SINCE my article on rickets was first set up in type, I have had the opportunity of examining a very interesting case, post mortem; it especially illustrates the deformities of the skull, the deformities of the chest, and the albuminoid condition.

A little girl was seen for the first time on April 20, 1868, when she was eighteen months old. Symptoms of rickets had begun to show themselves a year before; the head sweated very much; crusta lactea followed, and lasted several months. In January 1868, her mother observed that the sagittal suture was open.

State on April 20: Rickety to a second degree; emaciated; had several epileptiform attacks a day or two ago, no fits previously. Head certainly not enlarged; of natural shape, neither rickety nor hydrocephalic; sagittal suture open for its whole length, edges of bones half an inch asunder; coronal and lambdoid sutures open, but to a less extent. Slight deviation of face to the right; constant rigidity of the left arm; legs unaffected.

The fits did not recur until June 15, when she had several. The separation of the sutures had rather diminished. Then she seemed to improve again, had no fits, the sutures nearly closed up. But on August 1, the fits recurred; signs of consolidation of the lungs were detected on the 10th, and on the 19th she died. Her arm remained rigid throughout.

Post-mortem Examination.—Arm quite relaxed; no adapted atrophy of muscles. Shape of head and of face, and proportion between them, quite natural. Great beading of the costochondral articulations; depression of the cartilages in front of the nodules, no depression of the ribs.

Tubercular meningitis with great ventricular effusion; masses of cheesy tubercle embedded in cortex of the brain; a large mass in the right thalamus opticus. Dense miliary tuberculosis of both lungs. Tubercular ulcer in cæcum.

The skull.—Shape of vault of cranium quite natural, no increased prominence of frontal tuberosities; anterior fontanelle one inch square. Pericranium and dura mater very firmly adherent along the sutures; can be thoroughly separated only by scraping. Frontal suture closed, having left no vestige. Coronal and lambdoid sutures closed. Sagittal suture partially open. The best notion of the remaining appearances of the calvaria will be afforded if I begin by a description of the parietal bones. The parietal tuberosities are well marked; the bone in these spots is hard and translucent. Passing away from the protuberances towards the sutures, the bone becomes thicker, more opaque, and of more cancellous structure. At about half an inch from the coronal, and an inch from the sagittal suture, the texture and colour of the bone change, the change being abrupt and marked by an irregular line, which, there can be no doubt, indicates a former sutural margin of the bone. Beyond this line the bone is suddenly and considerably thickened for a space of from a quarter to half an inch, so that there is a well-marked low ridge or welt running round the bone; the osseous texture of this welt looks whiter and more compact than the other parts of the bone. This ridge is limited on its farther side, towards the sutures, by another line which exhibits very distinct remains of its former sutural character. Beyond this second line the bone becomes thinner, more succulent, and much more transparent; there is from a quarter to half of an inch of this bone on each side of the sutures and of the anterior fontanelle. The two parietal bones are in contact and well interdigitated for about half the length of the sagittal suture, but for the rest of its length, at the middle, the suture is open, a quarter of an inch across, and the space filled with membrane identical with that of the fontanelle. Edges of bones quite thin. Thus the bone has three distinct characters in different places, and these differences correspond, there can be little doubt, to three distinct stages of ossification:—

First, the stage of natural ossification, comprising the bone from its tuberosity to the first line of ancient suture.

Secondly, the stage of rickets; the bone being swelled, and in all respects in a condition like that of the enlarged ends of the ribs, and probably identical with it.

Thirdly, the stage of resumed natural ossification; comprising the bone between the second line of former suture and the real suture.

All these characters are much better marked on the outside than the inside of the bones; indeed they are hardly to be seen at all on the inside; which fact would seem to show that the pericranial ossification is much more deeply implicated by rickets than that formation of bone which proceeds from the dura mater.

On the dried skull the depression of the frontal suture is very well marked, although not great. The depression involves the area of resumed natural ossification.

It is particularly unfortunate that tubercular meningitis should have occurred to complicate this very interesting case. Otherwise we could have examined the state of brain and membrane which goes along, necessarily, with that separation of the sutures which is so hard to be understood in rickets. Is the opening of the sutures always associated with hydrocephalus? This we can neither affirm nor deny; yet more accurate post-mortem examinations are all that we need in order to settle that point. Opportunities are rather rare; twice within the present year have I examined cases of rickets with open sutures, and twice have I been thwarted in my wish to discover the real condition of the brain, free from all accidental complications. But let us assume that hydrocephalus is always present, is it a cause or an effect? Certainly I have always been inclined to look upon the effusion as secondary to the changes which take place in the growth of the skull and of the brain, and this because the condition of open suture is so easily recovered from; but then the objection comes with greater force, Why do the sutures open? Ritter regards the hydrocephalus as primary, and the cause of the open sutures; but then what causes the hydrocephalus? Virchow replies, the obstructed respiration and consequent venous stasis. I do not deny the truth of this explanation, but only say that it seems to me insufficient, and difficult of application in many cases.

I would draw attention to the fact that we proved, post mortem, that the depression of the cartilages in front of the thoracic beading was great, and that the depression of the ribs behind it was none.

Her spleen and kidneys were 'albuminoid,' her liver tough but fatty and opaque. The acute tuberculosis was the cause of her death, so that the albuminoid condition was altogether of second-rate importance. And indeed the child was gaining flesh until the acute tuberculosis set in, two or three weeks before death.

Frequency of Rickets.—In Virchow's 'Jahresbericht'* there is an abstract of a paper by Bruenniche, published last October in a Danish journal, giving the results of his investigations upon rickets as it exists in Copenhagen. Out of all sick children under five years of age seen by him, only 8·4 per cent. were rickety.† This suggests a contrast between Copenhagen on the one hand, and Prague, Dresden, and London on the other, which is very favourable to the former city; but when we find that the author observed only one case of the disease under six months of age, a doubt arises whether he has been sufficiently precise in his notion of rickets, and the doubt is strengthened by the stress which Bruenniche seems to lay upon bandy legs, a very tardy and (so far as diagnosis is concerned) unimportant evidence of rickets. With a view to the estimation of the influence of race upon the production of rickets, I tabulated all the sick children under five years of age, bearing unmistakable Irish names, whom I saw in 1867, and found that, out of forty-nine, twelve were rickety, or 24·5 per cent.

A paper by Scharlau upon congenital rickets, does not add hing positive to our knowledge of that subject.‡

ARTICLE XIX.—*A brief historical Sketch of the 'Abernethian'—some time known as the 'Medical and Philosophical'—Society of St. Bartholomew's.* By ROWLAND H. COOMBS.

MOST of our medical schools now have a Students' Society in connection with them for the reading and discussion of papers on medical and surgical subjects: these societies combine the distinctive features of the larger London medical societies. Our own, 'The Abernethian,' has existed, with varying success, but not under its present name, from the latter part of the eighteenth century, and its history, as I hope to show, is an interesting one. Although the students of our school have from time to time been made familiar with the names and deeds of its more distinguished members, yet, as no account of the Society has ever been published, this attempt to

* Bd. ii. S. 305. 1867.

† It is to be regretted that Ritter, Küttner, and Bruenniche, should have chosen the first five years of life as the basis of statistics; for there is no proof that rickets ever begins after the second year, and the signs of slight rickets tend to disappear rapidly as the health improves.

‡ 'Jahresbericht,' Bd. ii. S. 304. 1867.

supply the deficiency may be acceptable to past and future Bartholomew men. For the 'Abernethian' has been a part of the nurture ground of the staff of our Hospital and of the teachers of our school, and we cannot fail to notice how the officers and working members of the Society have become eminent as physicians and surgeons, not only in our own Hospital but in other London and provincial schools and in our universities.

The materials from which this history is compiled are of varying completeness: indeed, there are many years of which, the stability of the Society being very equivocal, no record is known to exist: therefore the different periods of the Society's history must needs be treated with unequal fulness.*

'The Medical and Philosophical Society of St. Bartholomew's' was founded by Mr. Abernethy in 1795; the 'Guy's Physical' was founded about 1775; but the oldest medical students' society is the 'Edinburgh Royal Medical,' which was founded in 1735: thus the 'Edinburgh Medical,' 'Guy's Physical' and the 'Abernethian' are older than the 'Medico-Chirurgical,' which was founded in 1805.

The object of the 'Medical and Philosophical' was, as now, the reading and discussion of papers on medical subjects, or such as had some direct bearing on medicine. When a paper or 'dissertation' (the title generally given to it in the Minutes) was not forthcoming, the narration of a case or the exhibition of morbid specimens filled up the gap. It had also a special object in view—the formation of a library. The 'Medical and Philosophical' was the common meeting ground for mutual advantage of past and present Bartholomew men, students, teachers, and old alumni. The 'Medical and Philosophical,' on the ruins of which the 'Abernethian' properly so called arose, was not entirely supported by students. At present the main feature of the 'Abernethian' is that it is chiefly a students' society, the staff of the Hospital and old pupils having only an occasional connection with it. It was not so with the 'Medical and Philosophical,' for Abernethy, who was connected with the Society from its foundation till his death, seemed to make it a part, so to speak, of his system of teaching.

It was not until the fifth session (1799–1800) that the Society appears to have begun to work regularly, and even then matters were far from being well ordered.

* My thanks are due to the present secretaries of the 'Abernethian' for the kindness with which they have placed at my disposal the records of the Society; also to Mr. Paget, and to many others, both present members of the school and old alumni, for valuable assistance and information.

The official staff of the Society consisted of six presidents, a librarian and treasurer (joint office), and a secretary. There was also a council, which appears to have had to discharge the duties which now belong to the committee of audit, and which also aided the other officers in managing the general business of the Society. In 1799–1800 (the fifth session) the six presidents were: Abernethy, Richard Powell, M.D., Joseph Hurlock, T. Bradley, M.D., James Macartney, William Blair. The librarian and treasurer was Mr. John Haslam and the secretary Mr. S. C. Hunt.

Before making any remarks on the session itself, I may be allowed to say a few words on some of the names given above; for, being the first names we meet with, they deserve, as far as possible, a somewhat detailed notice.

Of Abernethy I need only speak in connection with the Society. He was most regular in his attendance in the presidential chair, and contributed very largely to the stock of papers and cases; if there was a failure anywhere Abernethy appears to have invariably come forward and either started a conversational subject or narrated a case. He was especially ready to inform the members of any remarkable case that had occurred, or of any operation that had been done elsewhere; to report on papers which had been read to the Royal Society (of which he was then a Fellow) and to ask the opinion of the Society on the subject. His predilection for blue pill is well known, and he laid before the Society cases in which phthisis and hypochondriasis were much benefited by treatment on the mercurial plan. He gave papers on 'Tumours and their classification,' 'Hernia,' 'Ligature of Arteries'—in fact, to mention all the subjects which Abernethy brought before the Society for discussion would include the whole range of medical practice. He took great interest in the discussions on small-pox. It was just about this time (the exact date is 1798) that Jenner announced vaccination to the public and his statement was frequently the subject of debate.

Abernethy appears to have been in favour of inoculation by means of a thread saturated with matter, used as a seton.

Dr. F. Winslow says: 'Abernethy was one of the earliest friends of vaccination. In regard to the welcome with which "honest John," as Dr. Walker (for many years one of the presidents of the Society) used to call him, received this boon, we have the following particulars. The French apophthegm "Le scepticisme est le vrai flambeau de la science" ("Doubt is the true torch of science") was well sustained by Mr. Aber-

nethy when Jenner offered protection from the most contagious disease that ever desolated the earth. He listened with enthusiasm to the news, immediately corroborated and proclaimed by others; but of this wonderful and incomprehensible law in animal physiology he was sceptical; he waited for facts; it was only on witnessing them that he ceased to hesitate. He then zealously came forward in the formation and support of the vaccine institutions, and administered the guardian operation, the great prophylactic, to his own children.'

Richard Powell, M.D. (Oxon.), was elected physician to St. Bartholomew's in 1801, and held that office till 1824. 'He was esteemed a good chemist and was an active member of the committee for the revision of the 'Pharmacopœia Londinensis' of 1809, a translation of which he published.'

James Macartney, M.D., was afterwards Professor of Anatomy in the University of Dublin. 'He first described the process of healing by immediate union.' Macartney's immense and valuable collection of osteological and physiological preparations forms the major part of the museum which now enriches the university of Cambridge; but I should note, in passing, that the museum originated in a small number of preparations which were presented by Mr. Lawrence. Macartney contributed many papers and was a very prominent and useful member.

William Blair wrote some works on vaccination and on syphilis.

Mr. John Haslam (afterwards Dr. Haslam) was a noted man: he became distinguished as a successful practitioner in the department of mental disorders and also as a witness, in which capacity he was much sought after in all important disputed cases of insanity. He had a peculiar mannerism, 'and his mode of giving evidence, his acuteness and odd gesticulations often convulsed the court with laughter.' He was sometime resident physician to Bethlehem, and he read to the Society notes of cases which he had taken at that hospital. He published many works on insanity: his last work (1835) was entitled 'On the Nature of Thought or the Act of Thinking and its connection with a Perspicuous Sentence.'

It was during this session that the suggestion for the formation of a library arose; but the progress made was at first very slow and unsatisfactory. Each honorary member (these were members elected for writing specially worthy dissertations) contributed books to the value of one guinea, or subscribed one guinea; ordinary members paid a small subscription weekly. During subsequent sessions large numbers of books

were purchased, and presents of books were made by Abernethy, Macartney, Dr. Latham, and others. The library, if we may judge of it from the lists of books given in the Minutes, was an excellent and comprehensive one; and when the 'Medical and Philosophical' lapsed, the collection was added to the Hospital library. In 1805 the medical officers instituted a liberal annual donation to the funds, on condition that the books purchased therewith, as well as those previously in their possession, should be vested in the hands of trustees, who were to be the physicians and surgeons of the Hospital.

Up to the session 1801–1802 the Society was not very flourishing; the attendance of members was often so scanty as to compel an adjournment, and the doubtful practice of heavily fining members for non-attendance, or for failing to produce a paper when called on, was the cause of much contention. However, when for the session of 1801–1802, Mr. Lawrence was elected secretary, with James Macartney as librarian and treasurer, a good session was the result; no adjournments were necessary. Lawrence at this time was Abernethy's house-pupil, and both master and pupil worked energetically for the Society. Lawrence himself contributed largely in cases and also gave a long paper on the 'Function of the Skin,' which occupied several evenings in discussion. It may be as well to mention that in the Minutes of the Society at this period no abstract of 'papers' is ever given, only brief notices of the business done and of the cases discussed.

Another very active and useful member of the Society at this time, and who survived to 1867, was Mr. H. H. Carwardine. He came to London about two years later than Lawrence, who was his junior by about two years. 'A good surgeon, a good antiquarian, and a most kindhearted gentleman.'

Brodie was elected a member of the Society on November 3, 1801, the same year that he came to St. Bartholomew's. In his 'Autobiography'* Sir Benjamin says: 'During my first winter in London, I attended Mr. Abernethy's lectures on anatomy and worked in the dissecting room.' This will explain his connection with the Society, and that on January 5, 1813, the Minute says, 'Mr. Brodie's paper on "Animal Heat" was read to the Society by the president.' This paper is probably the one referred to in this passage from the 'Autobiography:' 'During the winter of 1810 and 1811 I communicated to the Society (the Royal) two physiological papers: one "On the

* Page 33.

Influence of the Brain on the Action of the Heart and the Generation of Animal Heat." . . . . They made a favourable impression at the time, so much so that the council awarded me the Copley medal in the autumn of 1811. At this time I was only twenty-eight years of age.' He does not appear to have maintained any intimate connection with the 'Medical and Philosophical,' yet the fact of his having had thus much to do with the Society should not be passed over unnoticed.

In the session of 1802–1803 Mr. Vincent was one of the presidents, Lawrence and Macartney still holding their former offices. Vincent was afterwards surgeon to the Hospital, and was esteemed one of the best practical surgeons of his time. Macartney, Carwardine, Lawrence, and Abernethy made frequent contributions, and this was doubtless one of the most successful sessions, as there was a balance in the hands of the treasurer, whereas previously there had been a deficit.

For the session 1803–1804 Lawrence contributed a paper 'On the Varieties of the Human Species,' thus foreshadowing his later views on comparative anatomy and kindred subjects; he also, in this and subsequent sessions, furnished many reports on cases of hernia. It was in the year 1806 that he gained the Jacksonian prize for an essay on hernia. His well-known book was based on this essay on hernia, and is now a standard classical work on the subject. During this session, too, we notice the name of Samuel Cooper as a contributor on the 'Treatment of fractured Thighs.' Cooper was the author of the well-known 'Surgical Dictionary,' and afterwards became surgeon to University College Hospital, having been defeated at St. Bartholomew's by the late Mr. Lloyd.

In the following session, 1804–1805, there is not much to note. The principal contributors were Abernethy, Lawrence, and Cooper: the latter read a paper on 'Cataract,' which was afterwards published.

In the session of 1805–1806, Langstaff (of whom it is said that he was 'the most zealous of museum collectors; he collected on morbid anatomy everything that was possible for him; his museum, he used to say, cost him 20,000*l.*, and when he was obliged to sell it he got less than 1,000*l.* for it') was an active working member; even so late as the year 1838 his name is to be found in the Minutes, and most frequently as an exhibitor of morbid specimens.

In the following session John Syer gave frequent cases and papers on the diseases of children, a subject on which he afterwards wrote.

Lawrence, in the session 1807–1808, read an elaborate paper on the 'Structure and formation of the Teeth.'

The name of Henry Earle, too (son of Sir Henry Earle, surgeon to the Hospital)—known as the inventor of Earle's fracture-bed, 'and in his time one of the surgeons most esteemed in private practice, and most popular among his pupils'—occurs frequently as a contributor of papers and cases.

Passing over the session of 1808–1809, as presenting nothing particularly noteworthy, we come to that of 1809–1810, when Mr. J. H. James, of Exeter, who is still living, was secretary. Mr. James 'was always distinguished as one of the best writers and teachers among the provincial surgeons.' During this session Mr. Joseph Hodgson, F.R.S., formerly surgeon to the Birmingham General Hospital, was elected a member, and besides the narration of cases of aneurism, he read a paper on 'Aneurism,' a subject which became a special study with him, and we may fairly look on his contributions here as the nucleus of his celebrated work entitled 'A Treatise on the Diseases of the Arteries and Veins.'

The name of Edward Stanley first appears as a contributor during this session.

In the next session, 1810–1811, Mr. Hodgson read another paper on the operation for aneurism and the application of the ligature. The Minute says: 'Mr. Hodgson was unanimously elected an honorary member, and the thanks of the Society were voted to that gentleman for his two papers on 'Aneurism.' We should notice, too, the name of a celebrated provincial surgeon, Mr. Charles Mayo, of Winchester, as the contributor of a paper on 'Erysipelas,' and also of reports of cases.

Mr. Stanley became secretary for the session 1811–1812, and distinguished himself by his energy and the prosperous manner in which he conducted the general business of the Society, and by the numerous cases he furnished for discussion. The Minutes of this session record the following circumstance: 'Mr. Earle submitted a proposition to the Society that the sum of twenty guineas be voted to Mr. Lawrence, in testimony of their esteem, and in acknowledgment of his exertions in forming and preserving the library. This motion was seconded by Mr. James, and carried unanimously.' And in the Minutes of the next meeting it is stated that Mr. Lawrence returned thanks to the Society for the donation voted him.

Dr. Peter Mere Latham, formerly physician to St. Bartholomew's, and justly famed as a writer and as an accomplished physician, was on the council this session.

During the session 1813–1814 Mr. Stanley resigned the post of secretary, and to note the value which was attached to his services he was unanimously elected an honorary member; but although he resigned his secretaryship he continued to act energetically for the Society, often, in the absence of a president, occupying the chair. After this time the Society began to decline, and was compelled to frame laws to ensure a better supply of papers; and although at this time Dr. Clement Hue (afterwards physician to the Hospital, and whose Harveian oration was much admired for its pure Latinity) and Dr. Gooch, the celebrated midwifery teacher, were among the presidents, and aided the Society much, yet at the end of the nineteenth session (1814–1815), it was agreed, 'that the meetings of the Society be but once a fortnight, and then on the Tuesdays on which the Medico-Chirurgical does not meet.'

William Kingdon at this time supported the Society continuously and energetically by filling the president's chair, and supplying cases and papers; and he continued to serve the 'Medical and Philosophical' up to the time of Abernethy's death; for when, in consequence of Abernethy's illness, the meetings were discontinued, William Kingdon was its president; so that to him belongs the honour of holding the post of last president of the 'Medical and Philosophical.' After this time, even so late as 1850, he gave his help in debate at the 'Abernethian.'

Kingdon, when a student, read a paper entitled 'Observations and Enquiries as to the probable Advantages to be derived from a frequent use of Blue Pill.' Abernethy himself was in the chair. The discussion lasted for two meetings, not so much on account of the merits of the paper, as of the opportunity thus given for discussing the wider subject of which Abernethy was, here and elsewhere, the frequent promoter—the constitutional treatment of all diseases, whether general or local.

Any detailed account of the period from 1815 to 1832 is, as before hinted at, impossible, since no authoritative record of the Society is known to exist; during the latter part of this period, Mr. Skey and Mr. Thomas Wormald, our consulting surgeons, and Mr. George Macilwain, the author of 'Memoirs of John Abernethy,' held office.

In 1826 Mr. Richard Owen read two papers—one 'On Encysted Calculus of the Urinary Bladder,' and the other 'A case of Gluteal Aneurism with ligature of the Internal Iliac.' The year previous to this Owen had been Abernethy's

prosector, and subsequently (being succeeded as demonstrator of anatomy by Messrs. Skey and Wormald) became the first occupier of the chair of comparative anatomy and physiology in our school. This professorship was specially created for him, and among his first pupils were Dr. Arthur Farre, Professor Rymer Jones, and Mr. White Cooper.

The 'Medical and Philosophical' held on till 1830, when, in consequence of the illness of Mr. Abernethy, the meetings were entirely discontinued; but in 1832 the Society was reorganised, and took the title of the 'Abernethian,' thus rendering a just tribute to the memory of one who had been its most constant and perhaps its most distinguished supporter.

'At a meeting held November 23, 1832, in the reading-room of St. Bartholomew's Hospital . . . it was resolved that a society of the medical pupils of this Hospital be founded, and be called the "Abernethian Society," and that the gentlemen present be enrolled as the founders of the same.' About thirty names are given, and among them we find the late Mr. Andrew Melville, Mr. McWhinnie, also Mr. Carsten Holthouse, now surgeon to the Westminster Hospital.

McWhinnie was well known as an accomplished anatomist, and was for some years assistant-surgeon to St. Bartholomew's. He read notes of cases of poisoning, and afterwards, in conjunction with Dr. Roupell, published a book entitled 'Illustrations of the Effects of Poisons.'

The official staff was the same as that of the 'Medical and Philosophical.' The six presidents were elected to serve one month each, during the six months of the winter session. It appears to have been the rule to elect the three house surgeons (for at this time St. Bartholomew's had only three full surgeons) with three other members as presidents; and the number was kept up to six until the session of 1849–1850, when an alteration was made by electing two presidents and four vice-presidents. Mr. Coote and the late Dr. Wm. Baly were the first two presidents.

The late Dr. Jeaffreson, Dr. Arthur Farre, Dr. Paget, of Cambridge, Mr. Spencer Smith, now surgeon to St. Mary's Hospital, Mr. John Gay, and the late Dr. Wm. Baly, are the names most frequently met with in the history of the first two or three years of the newly-constituted Society.

Undoubtedly one of the most original and noteworthy communications made to the 'Abernethian,' was the discovery of the 'Trichina Spiralis,' by Mr. Paget. The minute-book says: 'At a general meeting of the members of the Abernethian

Society, held February 6, 1835, Dr. Arthur Farre in the chair. . . . Mr. James Paget' (then a first year's student) 'favoured the Society by laying before it some drawings of a peculiar species of entozoön occurring in the human muscles. . . . The occurrence of these entozoa was by no means uncommon; they had been observed in one subject before this season, and Mr. Wormald had seen altogether twenty or thirty; hitherto, however, they had been regarded as small spicula of bone.' These quotations may be supplemented by reference to the letter of Mr. Paget, which appeared in the 'Lancet' for March 10, 1866.

In the session of 1834–1835 we find papers by Charles West on the 'Structure and Functions of the Placenta,' and, later on, among others of his, 'On the difficulties in the diagnosis and treatment of Head affections in Children.' Dr. West's fame as a writer and practitioner in a special department of medicine needs no comment, further than to remark that the line of practice in which he has become distinguished appears to have been marked out by his papers at the Abernethian. Mr. Spencer Smith was an active and able secretary, and his name, with those of Mr. Square, of Plymouth (author of papers on 'Affections of the Eye' and 'Hernia Cerebri'), Mr. John Gay, Mr. W. Acton, Mr. W. B. Kesteven, Mr. Holden, Mr. Coote, Mr. W. P. Ormerod, and Mr. Firth, of Norwich—all names of note in the profession—did good service between 1835 and 1840. Mr. Lloyd read papers on 'Scrofula,' a subject on which he wrote more fully in a work which gained the Jacksonian prize.

In the session of 1841–1842, Mr. C. H. Moore, one of the surgeons to the Middlesex Hospital, was secretary, and continued to hold office during the following session, and was afterwards president. Mr. Haynes Walton, well known as an ophthalmic surgeon, is now met with as president. We notice some valuable and original papers by Dr. G. Murray Humphry, the present Professor of Anatomy in the University of Cambridge, 'On the effects of Caries of the Vertebræ on the Spinal Cord,' and 'On the effects of Pressure on Bone.' Mr. Holmes Coote, an authority on syphilis, in 1842 communicated a paper on 'Primary Venereal Sores.' We note, too, in the session of 1842–1843, papers and cases by Dr. Edward L. Ormerod (at one time Demonstrator of Morbid Anatomy at St. Bartholomew's) on 'Fever;' also papers by the late Dr. Kirkes, Mr. T. W. Crosse, of Norwich, afterwards secretary, and Mr. Hussey, of Oxford, afterwards president.

Up to the commencement of the session 1844–1845, it had been the custom to begin the business of each session without any formality, but this session was inaugurated with an introductory address, 'On the advantages of Medical Societies,' by Mr. Paget; and from this time onward it has been the invariable rule for some officer of the school or Hospital, or for one of the presidents, or some senior member of the Society, to introduce the advantages accruing to students, and especially to the freshmen of the Hospital, by enrolling themselves amongst its members.

Mr. Mitchell Henry (formerly surgeon to the Middlesex Hospital) was president during the session of 1847–1848—a year of great financial prosperity—and the Society marked it by investing fifty pounds in the purchase of a president's chair.

For the session 1847–1848, Mr. John Abernethy Kingdon was secretary, and for the two following years, Mr. C. R. Thompson, of Westerham.

In the session 1849-1850, the laws of the Society were subjected to extensive revision and alteration, and besides the change before referred to in the number of presidents, one of the most important new laws related to the 'papers' read before the Society. It was determined that all 'papers' read before the Society should become its property, and that the more meritorious ones should be published under the superintendence of a committee. This 'Publishing Committee' (consisting of Dr. Baly, Mr. Paget, Mr. Coote, and Mr. T. M. Girdlestone), at the close of this session selected certain papers for publication in the 'Transactions.' The 'Transactions' were issued regularly until 1856, when, owing to the want of funds for the purpose, and from the obvious difficulty of selecting only a few for the honour, the practice was discontinued. Such papers as were not published in the 'Transactions,' at this period and subsequently, were copied, and retained in the Society's possession, and now form a valuable volume under the title of 'Unpublished Papers.'

Dr. Baly was president during 1849–1850; at his death, early in 1861, he had been connected with the Society for twenty-seven years; he succeeded to the office of treasurer in 1854, on the death of Dr. Roupell. Baly appears to have rendered great aid to the Society in debate, his great experience and pathological research constituting him on many points (e.g. cholera) an authority to which the members could appeal for a sound opinion.

We now come to a period, 1852–1853, of which we must

write briefly and inclusively. This year, when Harvey Ludlow, who died whilst serving in the Crimea, held office as secretary, was a flourishing one, aided as he was by Mr. H. Power, Mr. Savory, Mr. Callender, Mr. Jonathan Hutchinson (who contributed a paper 'On the forms of Dyspepsia which precede Phthisis'), Dr. H. Dobell, and later on by Dr. Edwards, Dr. Rolleston and Mr. T. Smith. All these contributed papers of value and interest.

To pursue the personal history of the Society further than this would be trespassing on the sensitiveness of many who now take an active part in its conduct, and it will therefore be better to stay our hand ten years short of the present date, leaving the pleasing task of making further additions to this sketch to some future member of our Society in long-distant years.

ARTICLE XIX.—*The Hospital Statistics.*

TABLE I.

SHOWING THE WHOLE NUMBER OF MEDICAL CASES UNDER TREATMENT, WITH THE RESULTS.

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER I.								
1. Smallpox	8	...	...	8	...	...	...	Removed to Smallpox hospital.
2. Chicken-pox	4	4	...	...	...	...	...	
3. Measles	3	3	...	...	...	...	...	
4. Scarlatina	28	28	...	...	...	...	...	
(*a*) Dropsy after Scarlatina	19	11	...	1	7	36·84	1·91	3 died within 24 hours after admission; 1 with pleuritic effusion, was tapped twice, died.
5. Diphtheria	4	2	...	...	2	50	·54	1 tracheotomy, died.
(*a*) Sequelæ of ditto	1	1	...	...	...	...	...	
6. Quinsy	14	14	...	...	...	...	...	
7. Croup	17	10	...	1	6	35·29	1·64	Tracheotomy in 5 cases; all died.
8. Continued Fever	7	5	...	...	2	28·57	·54	
(*a*) Typhus	39	33	...	...	6	15·38	1·64	
(*b*) Typhoid	61	47	...	...	14	20·30	40·3	1 death from perforation under five years; 2 from hæmorrhage of bowels.
(*c*) Febricula	39	37	...	2	...	...	...	
9. Hooping-cough	5	4	...	...	1	50	·27	
— Mumps	1	1	...	...	...	...	...	
10. Diarrhœa	24	22	...	2	...	...	...	
11. Dysentery	13	7	1	1	4	30·76	1·09	
12. Cholera	...	...	...	...	...	...	...	
(*a*) Choleraic Diarrhœa	1	1	...	...	...	...	...	
13. Catarrh	5	5	...	...	...	...	...	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER I.—*continued.*								
(*a*) With Aphonia	4	3	1	...	...	...	...	
15. Ague	7	7	...	...	...	...	...	
16. Erysipelas	29	25	1	1	2	6·89	·54	
18. Pyæmia	3	...	...	...	3	...	·82	1 case following necrosis of tibia, 1 degeneration of placenta in third month.
21. Rheumatism—								
(*a*) Acute	107	105	...	2	...	...	...	
(*b*) Subacute	39	38	...	1	...	...	..	
(*c*) With Cardiac Complication	39	37	...	1	1	2·56	·27	
(*d*) With Pericarditis	16	15	...	...	1	6·24	·27	3 cases complicated with pleuro-pneumonia and pericarditis, recovered.
— Muscular Rheumatism	5	4	...	1	...	...	...	
(*a*) Stiff-neck	4	4	...	...	...	...	...	
(*b*) Lumbago	3	3	...	...	...	...	...	
— Gonorrhœal Rheumatism	2	2	...	...	...	...	...	
— Synovial Rheumatism	8	8	...	...	...	...	...	
— Chronic Rheumatism	36	32	2	2	...	...	...	1 case complicated by iritis, recovered.
(*a*) Chronic Osteo-Arthritis	4	3	1	...	...	...	...	
24. Syphilis—								
(*a*) General	7	7	...	...	...	...	...	
(*b*) Hereditary	...	...	...	...	...	...	...	
26. Purpura	7	7	...	...	...	...	...	1 case complicated by pneumonia.
27. Scurvy	2	2	...	...	...	...	...	
32. Worms	1	1	...	...	...	...	...	
33. Hydatids	4	3	1	...	...	...	...	1 case H. of liver, tapped, opened, suppurated, healed.

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER II.								
1. Gout	3	2	...	...	1	33·33	·27	1 case complicated by hæmaturia.
2. Dropsy	5	1	...	1	3	20	·82	
— Amyloid Disease	2	2	...	...	...	...	...	
— Exophthalmic Goitre	2	1	1	...	...	...	...	
3. Anæmia	8	7	...	...	1	12·50	·27	
4. Cancer of Internal Organs	43	7	7	4	25	58·13	41·6	2 of œsophagus, death in one by perforation of aorta; 9 of stomach and pylorus; 1 of duodenum and pancreas; 2 of bronchial glands; 1 perforating pulmonary artery; 1 of left lung and adjacent ribs; 1 of head of humerus; 14 of uterus.
— Leucocythæmia	1	...	...	...	1	...	·27	
9. Tumours	7	6	...	...	1	14·28	·27	
14. Scrofula	8	6	1	1	...	...	...	
15. Psoas Abscess	1	1	...	...	...	...	...	
17. Tuberculosis	7	...	...	...	7	...	1·91	1 case of tubercle in pericardium and upon heart's substance.
(*a*) Tubercular Peritonitis	...	...	...	...	...	...	...	
(*b*) Ditto Meningitis	4	...	...	...	4	...	1·09	
(*c*) Tubercle in Brain	1	...	...	...	1	...	·27	
18. Phthisis	178	85	15	...	78	43·72	21·36	Complicated by hæmoptysis in 18 cases, of which 5 died; by hæmorrhage from lungs in 2 cases, which died; by hæmorrhage from bowel in 1 case, which died; by fistula in 1 case, relieved; by diarrhœa in 6 cases, all fatal.

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER II.—*continued.*								
19. Hydrocephalus	1	...	...	1	...	...	...	
ORDER III.								
1. Meningitis	1	...	...	...	1	...	·27	
2. Encephalitis	...	...	...	...	...	...	...	
(*a*) Acute Softening	1	...	...	...	1	...	·27	
(*b*) Chronic ditto	2	...	2	...	...	...	...	
(*c*) Cerebral Abscess	3	...	...	...	3	...	·82	
4. Apoplexy	13	...	1	...	12	92·30	3·28	1 aneurism of cerebral artery, plus morbus cordis.
5. Paralysis	4	3	...	...	1	25	·27	
(*a*) Hemiplegia	30	27	...	1	2	66·66	·54	Right H. with affected speech, 10; unaffected, 2. Left H. with affected speech, 2; unaffected, 16.
(*b*) Paraplegia	21	16	1	1	3	14·28	·82	1 disease of Atlo-articulation, pressure, death.
(*c*) Locomotor Ataxy	1	...	1	...	...	...	...	
(*d*) Progressive Muscular Atrophy	2	1	1	...	...	...	...	
— Local Paralysis	2	2	...	...	...	...	...	
(*a*) Facial Palsy	...	...	...	...	...	...	...	
6. Paralysis Agitans	2	1	1	...	...	...	...	
7. Chorea	36	35	...	1	...	...	...	
8. Epilepsy	67	61	2	1	3	44·92	·82	
(*a*) Epileptic Vertigo	2	2	...	...	...	...	...	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER III.—*continued.*								
9. Tetanus	...	...	...	...	...	...	...	
(*a*) Idiopathic	1	1	...	...	...	...	...	
10. Hysteria	27	21	2	4	...	...	...	
11. Convulsions	3	2	...	...	1	33·33	·27	
(*a*) Spasm of Muscles	2	2	...	...	...	...	...	1 posterior neck muscles, 1 glosso-pharyngeal muscles.
12. Insanity	...	...	...	...	...	...	...	
(*a*) Hypochondriasis	1	1	...	...	...	...	...	
14. Neuralgia	3	3	...	...	...	...	...	
(*a*) Facial	...	...	...	...	...	...	...	
(*b*) Hemicrania	2	2	...	...	...	...	...	
(*c*) Pleurodynia	4	4	...	...	...	...	...	
(*d*) Sciatica	11	9	1	1	...	...	...	
16. Disease of Brain	4	2	...	1	1	25	·27	Death from syphilitic disease of dura mater, secondary softening.
(*a*) Mania	...	...	...	...	...	...	...	
(*b*) Nervous Debility	1	1	...	...	...	...	...	
17. Delirium Tremens	17	13	...	...	4	23·52	10·9	
18. Disease of Spinal Cord	1	...	...	...	1	...	·27	
ORDER IV.								
2. Pericarditis	5	1	...	...	4	80	10·9	
5. Disease of Heart	26	20	2	...	4	15·38	38·9	1 case of myo-carditis.
(*a*) Valvular	20	10	5	...	5	25	13·6	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER IV.—*continued.*								
5. Disease of Heart—								
(*b*) Cardiac Dropsy . . .	29	2	2	...	25	86·20	41·6	
6. Aneurism of Aorta . . .	10	2	1	1	6	60	16·4	
— Dilatation of Aorta . . .	...	...	...	...	...	...	...	
— Congenital Malformation of Aorta .	1	...	...	...	1	...	·27	
7. Aneurism of other Arteries . .	3	...	1	...	2	66·66	·54	1 of innominata artery, 1 of superior mesenteric artery.
8. Angina Pectoris	1	...	...	...	1	...	·27	
11. Phlebitis	2	2	...	...	...	...	...	
(*a*) Phlegmasia dolens . . .	3	3	...	...	...	...	...	
ORDER V.								
2. Hæmoptysis	20	18	...	1	1	50	·27	
3. Laryngitis	...	...	...	...	...	...	...	
5. Bronchitis	30	24	...	3	3	10	·87	
(*a*) Acute	8	2	...	...	6	75	16·4	
(*b*) Capillary	9	5	...	...	4	44	10·9	1 complicated by cancrum oris, died.
(*c*) Chronic, with Emphysema .	32	18	1	1	12	37·50	32·8	3 cases of anasarca with albuminuria; 1 of fatty heart, died.
6. Pleurisy	15	15	...	...	...	...	...	
(*a*) Simple Effusion . . .	16	12	...	...	4	80	1·09	
(*b*) Purulent Effusion . . .	2	1	1	...	...	...	...	
7. Congestion of Lungs . . .	...	...	...	...	...	...	...	
8. Pneumonia	13	12	...	...	1	76·92	·27	
(*a*) Right Lung	2	1	...	...	1	50	·27	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER V.—*continued.*								
8. Pneumonia—								
(*b*) Left Lung	9	6	...	...	3	33·33	·87	
(*c*) Double	...	...	...	...	...	...	...	
9. Pleuro-pneumonia	21	13	...	...	8	38	2·19	2 with pericarditis, recovered; 1 with gangrene, died.
10. Asthma	1	1	...	...	...	...	...	
12. Disease of Lungs	...	...	...	...	...	...	...	
(*a*) Cirrhosis	1	...	...	...	1	...	·27	
(*b*) Gangrene	1	...	...	...	1	...	·27	
ORDER VI.								
1. Stricture of Œsophagus	1	1	...	...	...	...	...	
3. Stomatitis	1	1	...	...	...	...	...	
4. Gastritis	...	...	...	...	...	...	...	
(*a*) Chronic ditto	1	1	...	...	...	...	...	
— Ulcer of Stomach	3	2	...	...	1	33·33	...	1 perforation, peritonitis, died.
5. Enteritis	...	...	...	...	...	...	...	
(*a*) Typhlitis	6	5	...	...	1	16·66	...	1 perforation, peritonitis, died.
7. Peritonitis	2	1	...	...	1	50	...	
(*a*) Acute	...	...	...	...	...	...	...	
(*b*) Chronic	1	...	...	...	1	...	...	
8. Constipation	29	27	...	...	2	68·96	·54	1 with colonitis and peritonitis, died.
— Obstruction	1	...	...	...	1	...	...	Perforation, peritonitis, death.
9. Intussusception	...	...	...	...	...	...	...	
10. Stricture of Intestines	2	1	...	...	1	50	...	1 case stricture of sigmoid flexure, probably carcinomatous.

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER VI.—*continued.*								
10. Stricture of Intestines—								
(*a*) Intestinal Strangulation	1	...	...	...	1	...	...	Perforation, peritonitis; death.
(*b*) Twisting of Gut	1	...	...	...	1	...	...	Twist of colon on itself, 4 days, death.
11. Ulceration of Intestines	...	...	...	...	...	...	...	
14. Dyspepsia	11	9	1	1	...	...	...	
(*a*) Gastrodynia	1	1	...	...	...	...	...	
15. Pyrosis	2	2	...	...	...	...	...	
16. Chronic Vomiting	6	4	...	2	...	...	...	
17. Hæmatemesis	16	15	...	...	1	62·50	·27	
18. Melæna	5	4	...	1	...	...	...	
19. Hæmorrhoids	3	3	...	...	...	...	...	
22. Disease of Spleen	1	1	...	...	...	...	...	
(*a*) Congestion	...	...	...	...	...	...	...	
(*b*) Hypertrophy	3	2	1	...	...	...	...	
23. Disease of Liver	3	2	...	...	1	33·33	...	
(*a*) Enlargement	8	3	1	1	3	37·50	·87	1 case of enlarged spleen and œdema of lung, died.
24. Jaundice	9	8	...	1	...	...	...	
25. Hepatitis	...	...	...	...	...	...	...	
— Hepatic Abscess	1	...	...	1	...	...	...	
26. Cirrhosis	8	4	1	...	3	37·50	·87	Ascites in 1 case, paracentesis, death.
27. Gallstones	1	1	...	...	...	...	...	
28. Ascites (Hepatic)	8	4	...	...	4	50	1·09	
29. Colic	...	...	...	...	...	...	...	
30. Lead Poisoning	...	...	...	...	...	...	...	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER VI.—*continued.*								
30. Lead Poisoning—								
(*a*) Lead Colic	19	19	...	...	...	...	...	
(*b*) Lead Palsy	3	3	...	...	...	...	...	
ORDER VII.								
— Disease of Supra Renal Capsules	1	...	...	...	1	...	·27	
1. Nephritis (Acute Renal Disease)	14	3	...	1	10	71·42	2·73	1 complicated with phthisis; 3 deaths from effusion into both pleuræ, 1 from pericarditis plus ulcerated aortic valves.
2. Albuminuria (Chronic Renal Disease)	59	28	3	2	26	44	7·12	Deaths by apoplexy, pleurisy, œdema of lung, bronchitis, and broncho-pneumonia, with effusion.
5. Diabetes	10	6	2	...	2	20	·54	
6. Disease of Kidneys	...	...	...	...	...	...	...	
(*a*) Renal Calculus	1	1	...	...	...	...	...	
(*b*) Abscess of Kidney	1	1	...	...	...	...	...	In or about left kidney.
7. Hæmaturia	3	1	1	...	1	33·33	·27	1 complicated with phthisis.
9. Gravel	1	1	...	...	...	...	...	
10. Cystitis	1	...	1	...	...	...	...	
(*a*) Acute	1	1	...	...	...	...	...	
(*b*) Chronic	2	...	2	...	...	...	...	
ORDER VIII.								
4. Hæmatocele	6	6	...	...	...	...	...	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER VIII.—*continued.*								
7. Metritis	1	1	...	...	...	...	...	
(*a*) Acute	...	...	...	...	...	...	...	
(*b*) Chronic	19	19	...	...	...	...	...	
8. Ovarian Dropsy	3	1	2	...	...	...	...	
9. Ovarian Tumour	2	1	...	...	1	50	·27	1 tapped three times, died.
11. Uterine Tumour	...	...	...	...	...	...	...	
(*a*) Fibroid	18	14	3	1	...	...	...	
(*b*) Vascular Tumour	4	4	...	...	...	...	...	All removed by operation.
12. Uterine Polypus	7	7	...	...	...	...	...	6 removed by operation.
14. Vesico-vaginal Fistula	...	...	...	...	...	...	...	
16. Disorders of Menstruation	...	...	...	...	...	...	...	
(*a*) Amenorrhœa	7	6	1	...	...	...	...	
(*b*) Menorrhagia	3	3	...	...	...	...	...	
(*c*) Mechanical Dysmenorrhœa	11	10	...	1	...	...	...	Cervix divided in 5 cases.
— Chlorosis	3	2	...	1	...	...	...	
17. Disease of Uterus	1	...	1	...	...	...	...	
(*a*) Congestion	2	2	...	...	...	...	...	
(*b*) Subinvolution	3	3	...	...	...	...	...	
(*c*) Anteversion	4	4	...	...	...	...	...	Operation in 2 cases of anteversion.
(*d*) Retroversion	1	1	...	...	...	...	...	
(*e*) Procidentia	3	3	...	...	...	...	...	
(*f*) Elongated Cervix	6	6	...	...	...	...	...	
(*g*) Cicatrices of Cervix	2	2	...	...	...	...	...	
(*h*) Atrophy of Uterus	1	...	...	1	...	...	...	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER VIII.—*continued.*								
— Disorders of Pregnancy . . .	3	1	...	2	...	...	...	
— Uterine Hæmorrhage . . .	1	1	...	...	...	...	...	
— Pelvic Cellulitis	16	15	1	...	...	...	...	
— Puerperal Mania	1	1	...	...	...	...	...	
ORDER IX.								
2. Periostitis	2	2	...	...	...	...	...	
ORDER X.								
3. Abscess	...	...	...	...	...	...	...	
7. Urticaria	2	2	...	...	...	...	...	
8. Eczema	...	...	...	...	...	...	...	
(*a*) Acute	3	3	...	...	...	...	...	
(*b*) Chronic	...	...	...	...	...	...	...	
(*c*) General	1	1	...	...	...	...	...	
9. Herpes	...	...	...	...	...	...	...	
(*a*) Herpes Zoster	1	1	...	...	...	...	...	
12. Scabies	2	1	...	1	...	...	...	
(*a*) Pustular	1	1	...	...	...	...	...	
15. Ecthyma	1	1	...	...	...	...	...	
26. Erythema Nodosum . . .	5	4	...	1	...	...	...	
27. Gangrena Senilis	...	...	...	...	...	...	...	

TABLE I.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, with Remarks
ORDER XI.								
1. Intemperance	32	31	...	...	1	3·12	·27	
2. Privation	3	2	...	...	1	33·33	·27	
3. Neglect (Vermin)	1	...	1	...	...	...	...	
5. Malformations	...	...	...	...	...	...	...	
8. Debility	47	44	...	3	...	...	...	
(*a*) From Hyperlactation . .	2	2	...	...	...	...	...	
*9. Poison	15	13	...	...	2	13·33	·54	1 death from laudanum, 1 death from strychnine.
(*a*) Mercurial Poisoning . .	3	2	...	...	1	33·33	...	Death complicated by chronic bronchitis and amaurosis.
12. Drowning	1	1	...	...	...	...	...	
Diseases not classed	2	2	...	...	...	...	...	
Total	1938	1429	78	67	365			

* Of the poisoning cases liq. ammon. was taken in 1 case; strychnine in 2; sulphate of iron in 1; sulphate of zinc in 1; carbolic acid in 1; oxalic acid in 1; hydrochloric acid in 1; arsenic in 1; corrosive sublimate in 1; parafine oil in 2; ammonia in 1.

TABLE II.

SHOWING THE WHOLE NUMBER OF SURGICAL CASES UNDER TREATMENT, WITH THE RESULTS.

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, and Remarks
ORDER I.								
6. Quinsy	8	8	...	...	...	...	...	
16. Erysipelas—								
(*a*) Simple	22	21	...	...	1	4·54	·54	
(*b*) Phlegmonous	12	7	...	...	5	41·66	2·73	Pyæmia 3.
(*c*) Cellulitis	83	82	...	...	1	1·21	·54	Erysipelas 2; bronchitis 1.
(*d*) Elephantiasis	2	1	1	...	...	...	...	
18. Pyæmia—								
(*a*) Acute	2	...	...	...	2	100	1·09	
(*b*) Chronic	...	...	...	...	...	...	...	
24. Syphilis—								
(*a*) Primary	167	140	...	27	...	...	...	Phagedæna 2; erysipelas 1.
(*b*) Secondary	213	185	...	24	4	1·87	2·18	Erysipelas 4.
(*c*) Inherited	5	5	...	...	...	...	...	
25. Gonorrhœa, including Bubo and Phymosis	249	199	...	48	2	·80	1·09	Fever 2; smallpox 1; erysipelas 1; phagedæna 1.
27. Scurvy	...	...	...	...	...	...	...	
ORDER II.								
4. Cancer—								
(*a*) Of External Organs	65	38	16	3	8	12·30	4·37	Erysipelas 3; gangrene of wound 2; pyæmia 1.

TABLE II.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, and Remarks
ORDER II.—*continued.*								
6. Lupus	7	7	...	...	...	...	...	
8. Polypus	3	3	...	...	...	...	...	
9. Tumours	66	58	5	1	2	3·03	1·09	Pyæmia 2; phagedæna 1.
10. Mortification—								
(*a*) Primary	4	4	...	...	...	...	...	
(*b*) Secondary	4	2	...	...	2	50	1·09	
11. Phagedæna	...	...	...	...	...	...	...	
12. Bed-sores	1	1	...	...	...	...	...	
13. Fistula	26	23	1	...	2	7·69	1·09	Hæmorrhage 1; pyæmia 1.
14. Scrofula	14	9	4	...	1	7·14	·54	
15. Psoas Abscess, including Pelvic	23	15	1	...	7	30·43	3·82	Erysipelas 1.
ORDER III.								
9. Tetanus	6	3	...	...	3	50	1·63	
14. Neuralgia	12	12	...	...	...	...	...	
18. Disease of Spinal Cord	8	1	6	1	...	...	...	
19. Conjunctivitis	8	7	1	...	...	...	...	
20. Amaurosis	4	...	4	...	...	...	...	
21. Cataract	9	7	1	1	...	...	...	
22. Iritis	5	5	...	...	...	...	...	
23. Corneitis	12	12	...	...	...	...	...	
24. Disease of Eye	10	8	2	...	...	...	...	Erysipelas 1.
25. Disease of Ear	...	...	...	...	...	...	...	

TABLE II.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, and Remarks
ORDER IV.								
7. Aneurism	6	3	...	1	2	33·33	1·09	Axillary 1, subclavian artery tied. Femoral 1, external iliac tied. Femoral (traumatic) 1, femoral tied, sec. hæmorrhage, amp. thigh. Popliteal 3, femoral tied 1; amp. thigh for gangrene leg 1
11. Disease of Arteries. . . .	...	...	...	...	...	...	...	
12. Phlebitis	4	4	...	...	...	...	...	
13. Disease of Veins	25	23	1	1	...	...	...	22 complicated with ulcer or eruption.
(*a*) Rupture of do. . . .	3	2	...	...	1	33·33	·54	
14. Nævus	8	8	...	...	...	...	...	
ORDER V.								
1. Epistaxis	3	3	...	...	...	...	...	
3. Laryngitis	1	1	...	...	...	...	...	Tracheotomy.
ORDER VI.								
1. Stricture of Œsophagus . . .	5	1	2	...	2	40	1·09	Œsophagotomy 1.
3. Stomatitis	6	4	...	...	2	33·33	1·09	
(*a*) Glossitis	1	...	...	1	...	...	...	
10. Stricture of Rectum . . .	2	2	...	...	...	...	...	
11. Ulceration of do.	4	3	1	...	...	...	...	

TABLE II.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, and Remarks
ORDER VI.—*continued.*								
13. Hernia—								
(*a*) Femoral	22	13	...	...	9	40·90	4·89	Taxis 9. Pyæmia 1; apoplexy 1; sec. hæmor. 1.
(*b*) Inguinal	30	26	...	...	4	13·33	2·18	Taxis 26.
(*c*) Otherwise	3	1	...	...	2	66·66	1·09	Taxis 1 (umbilical).
19. Hæmorrhoids	15	15	...	...	...	...	...	
ORDER VII.								
7. Hæmaturia	1	1	...	...	...	...	...	
8. Stone	10	8	...	...	2	20	1·09	
10. Cystitis	13	10	2	1	...	...	...	
11. Disease of Prostate Gland	6	5	...	...	1	16·66	·54	
12. Stricture of Urethra, including Retention of Urine	45	42	...	...	3	6·66	1·63	
13. Extravasation of Urine, including Urinary Abscess	20	18	...	...	2	10	1·09	
14. Incontinence of Urine	2	1	1	...	...	...	...	
ORDER VIII.								
2. Orchitis	3	2	...	1	...	...	...	
3. Hydrocele	20	20	...	...	...	...	...	
4. Hæmatocele	1	1	...	...	...	...	...	
5. Disease of Testicle	7	5	2	...	...	...	...	Pyæmia 1.
9. Ovarian Tumour	13	6	1	...	6	46·15	3·27	
18. Disease of Female Organs (External)	17	13	4	...	...	...	...	Sec. hæmorrhage and phagedæna 1.

TABLE II.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, and Remarks
ORDER IX.								
1. Synovitis	73	68	3	...	2	2·75	1·09	Fever 1; pyæmia 1.
2. Periostitis	7	7	...	...	...	...	...	
3. Exostosis	4	3	...	...	1	25	·54	Erysipelas 1; pyæmia 2.
4. Caries	24	19	4	1	...	...	...	
5. Necrosis	91	69	12	2	8	8·51	4·37	Pyæmia 3; sunstroke 1.
6. Disease of Bone	4	2	1	...	1	25	·54	
7. Curvature of Spine	5	2	3	...	...	...	...	
8. Disease of Joints	119	83	21	6	9	7·56	4·80	Erysipelas 3; pyæmia 2.
(*a*) Disease of Glands	26	25	...	...	1	3·84	·54	Erysipelas 1.
(*b*) Disease of Bursæ, Ganglia, &c.	73	73	...	...	...	...	...	Dif. cellulitis 1.
ORDER X.								
1. Carbuncle	21	19	...	...	2	9·54	1·09	Erysipelas 1.
3. Abscess	121	119	1	...	1	·82	·54	Erysipelas 1; pyæmia 1; morb. Brightii 1.
(*a*) Chronic	9	8	...	...	1	11·11	·54	Erysipelas 1; pyæmia 2; bronchitis 1.
4. Ulcer	90	83	2	2	3	3·33	1·63	
5. Disease of Skin	1	1	...	...	...	...	...	
8. Eczema	12	11	1	...	...	...	...	Erysipelas 1.
12. Scabies	...	...	...	...	...	...	...	
13. Pemphigus	...	...	...	...	...	...	...	
18. Erythema Nodosum	4	4	...	...	...	...	...	
21. Psoriasis	...	...	...	...	...	...	...	
26. Gangrena Senilis	2	1	...	1	...	...	...	

TABLE II.—*continued.*

DISEASES	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes	Nature of Complications, and Remarks
ORDER XI.								
5. Malformations	64	55	4	4	1	1·56	·54	
10. Violence and Injury—								
(*a*) Burn and Scald	100	71	...	4	25	25	13·66	Tetanus 2; pneumonia 1.
(*b*) Contusions	206	195	...	...	11	5·34	6·01	Erysipelas 1; lacer. viscera 2.
(*c*) Sprains	49	47	...	...	2	4·08	1·09	Meningitis 1; pneumonia 1.
(*d*) Gunshot Wounds	2	1	...	...	1	50	·54	Suicide 1; pneumonia 1.
(*e*) Laceration of Limbs	11	10	...	...	1	9·09	·54	Lacer. viscera 1.
(*f*) Wounds	196	186	...	4	6	3·06	3·27	Erysipelas 4; cellulitis 6; hæmorrhage 1; tetanus 2; bronchitis 1.
(*g*) Concussion of Brain and Cord	52	47	...	3	2	3·85	1·09	Abscess brain 1; meningitis 1.
(*h*) Fractures—								
Simple	387	368	1	...	18	4·65	9·81	
Compound	58	50	...	...	8	13·79	4·37	
Ununited and Mal-union of	10	5	5	...	...	...	...	See Sub-Table.
(*k*) Dislocations—								
Simple	33	30	3	...	...	...	...	
Compound	2	1	...	...	1	50	·54	
13. Suicide (Cut Throat)	8	6	...	...	2	25	1·09	Dif. cellulitis 1.
Diseases not classed	20	18	2	...	...	...	...	
TOTAL	3232	2792	119	138	183	5·88		
Remaining January 1, 1868, 343.								

SUB-TABLE (FRACTURES AND DISLOCATIONS).

DISEASES	Under 5 years		5–10		10–15		15–25		25–35		35–45		45–55		55–65		65–75		75–85		Above 85		Total		Deaths		Per-centage of Deaths	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
SIMPLE FRACTURES—																												
Skull, Vault	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	2	..	..	..				
,, Base	..	..	..	1	..	..	1	..	4	..	2	..	1	..	1	1	..	1	..	..	9	3	4	2	44·44	66·66		
Spine, Cervical	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	1	..	100			
,, Dorsal	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	2	..	2	..	100			
,, Lumbar	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	100			
,, Sacrum and Coccyx	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..				
Bones of Face	..	..	..	..	..	1	..	..	2	..	1	..	1	..	..	..	..	..	..	..	4	1	..	..				
Sternum	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..				
Ribs	..	1	..	..	2	..	4	1	2	2	7	3	5	3	9	1	1	2	1	..	..	..	31	13	4	..	12·9	
Clavicle	..	..	2	..	1	1	1	..	2	1	3	..	2	..	1	..	..	..	..	..	..	..	12	2	..	..		
Scapula	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..		
Humerus	2	1	1	..	..	1	2	..	4	..	..	..	2	3	2	..	..	..	..	..	..	..	13	5	..	..		
Radius and Ulna	..	..	1	..	..	..	3	..	..	..	2	..	1	1	1	1	..	1	..	..	..	..	8	3	..	..		
Bones of Hand	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
Pelvis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Femur, Neck	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	1	4	..	3	1	1	3	10	1	2	33·33	20
,, Shaft	9	4	12	5	4	..	5	3	..	..	5	..	5	1	1	1	2	..	1	..	..	..	44	14	..	..		
Patella	..	..	..	..	..	..	2	1	6	..	6	2	3	4	..	1	2	..	..	1	..	..	19	9	..	..		
Tibia and Fibula, or Tibia alone	2	2	5	1	3	1	19	3	21	3	23	2	15	6	4	2	5	2	..	..	..	1	97	23	..	1		4·34
Fibula alone	..	..	2	..	1	..	10	2	9	3	12	3	8	2	6	1	1	..	..	..	..	..	49	11	..	..		
Bones of Foot	..	..	..	..	..	..	2	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..		
TOTAL	13	8	23	7	12	4	49	10	55	10	64	10	45	20	26	10	12	10	2	4	1	2	302	95	..	..		
Deduct Multiple Fractures of separate Bones																							8	2	..	..		
Total Patients under Treatment																							294	93	13	5	4·42	5·37

SUB-TABLE (FRACTURES AND DISLOCATIONS)—*continued.*

DISEASES	Under 5 Years		5–10		10–15		15–25		25–35		35–45		45–55		55–65		65–75		75–85		Above 85		Total		Deaths		Per-centage of Deaths	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
COMPOUND FRACTURES—																												
Skull, Vault	1	..	1	..	..	..	2	..	1	..	1	1	1	..	..	..	1	..	..	..	..	..	8	1	4	..	50	
Face	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
Clavicle and Scapula	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Sternum and Ribs	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Humerus	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Radius and Ulna	..	..	..	..	..	..	2	..	..	..	1	..	..	..	2	1	..	..	..	..	..	..	5	1	1	..	20	
Bones of Hand	..	..	1	..	1	..	4	..	4	..	1	..	..	..	1	..	..	..	..	..	..	..	12	..	..	..		
Pelvis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Femur	..	1	1	..	..	..	2	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	4	1	1	..	25	
Patella	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
Tibia or Fibula	..	1	1	..	..	..	2	..	3	..	..	1	5	1	..	..	1	..	..	..	..	..	12	3	3	..	25	
Bones of Foot	..	..	..	..	1	1	4	..	1	..	5	..	1	..	..	..	..	..	..	..	..	..	12	1	1	..	8·33	
TOTAL	1	2	4	..	3	1	17	..	9	..	8	2	7	1	4	1	2	..	..	..	..	..	55	7	10	..		
Deduct Multiple of Fractures of separate Bones																							4	..	2	..		
Total Patients under Treatment																							51	7	8	..	15·69	
DISLOCATIONS, SIMPLE—																												
At Shoulder Joint	..	..	..	..	..	..	..	..	..	1	2	..	1	1	1	..	..	2	..	..	..	..	4	4	..	..		
„ Elbow „	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..		
„ Hip „	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..		
Of Patella	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
At Knee Joint	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
„ Ankle „	..	..	..	..	1	..	1	2	1	..	4	..	2	..	..	..	..	1	..	..	..	..	9	3	..	..		
„ Lesser Joints	..	..	1	..	..	..	1	..	1	1	2	..	..	..	..	..	1	..	..	..	..	..	6	1	..	..		
TOTAL	1	..	1	..	3	..	2	3	4	2	8	..	3	1	1	..	1	3	..	..	..	..	24	9	..	..		
DISLOCATIONS, COMPOUND—																												
At Elbow Joint	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
„ Wrist „	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	..		
„ Ankle „	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1	..	1		100
Of Digits	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
TOTAL	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	1	1	..	1		100

SUB-TABLE SHOWING TOTAL NUMBER OF CASES OF ERYSIPELAS, PYÆMIA, ETC.

DISEASES	Under 5 Years		5–10		10–15		15–25		25–35		35–45		45–55		55–65		65–75		75–85		Above 85		Total		Deaths		Per-centage of Deaths	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1. Erysipelas—Admissions	2	3	1	..	2	..	4	3	4	2	4	1	4	1	..	..	2	1	..	..	..	..	23	11	5	1	21·73	9·09
2. Do. occurring in Hospital	..	..	..	..	1	..	1	4	1	3	2	3	..	..	..	..	1	2	..	..	..	..	6	12	2	1	33·33	8·33
3. Do. do. after Operations	..	..	1	..	..	..	2	1	3	1	..	1	3	1	..	..	..	..	..	..	..	..	9	4	..	..		
1. Pyæmia—Admissions	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	2	..	100	
2. Do. occurring in Hospital	..	..	1	..	..	1	1	..	2	..	..	2	2	1	..	..	..	..	..	..	..	..	6	4	5	4	83·33	100
3. Do. do. after Operations	..	..	..	..	1	..	2	1	3	1	1	1	1	..	..	..	..	1	..	..	..	..	8	4	7	3	87·5	75
1. Phagedæna—Admissions	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Do. occurring in Hospital	..	..	..	..	..	..	2	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	2	2	..	..		
3. Do. do. after Operations	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..		
1. Gangrene—Admissions	..	1	..	..	..	..	1	..	1	1	2	..	..	..	..	..	1	1	..	..	..	..	5	3	1	1	20	33·33
2. Do. occurring in Hospital	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
3. Do. do. after Operations	..	..	..	..	1	..	..	..	..	1	..	..	1	1	..	..	..	1	..	..	..	..	2	3	..	1		33·33
1. Diffuse Cellulitis—Admissions	..	..	2	..	2	1	11	3	10	8	15	6	10	4	6	4	..	1	..	..	..	..	56	27	..	1		3·70
2. Do. occurring in Hospital	..	..	..	..	1	..	1	..	..	..	3	1	2	..	1	..	1	..	..	..	..	..	9	1	1	..	11·11	
3. Do. do. after operations	..	..	..	..	..	1	1	..	3	1	1	..	..	1	..	..	..	..	..	..	..	..	5	3	..	..		
1. Tetanus—Admissions	..	..	..	..	1	..	..	..	2	..	2	..	1	..	..	..	..	..	..	..	..	..	6	..	3	..	50	
2. Do. occurring in Hospital	..	..	..	..	..	..	1	1	..	..	2	1	..	..	..	..	..	..	..	..	..	..	3	2	2	2	66·66	100
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Fevers—occurring in Hospital	..	..	1	..	..	..	..	5	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2	5	..	2		40
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Rheumatism—occurring in Hospital	..	..	..	..	..	..	..	..	2	1	..	..	1	..	1	..	..	..	..	..	..	..	4	1	..	..		
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Œdema Glottidis, Croup, Bronchitis, Pleurisy, Pneumonia—occurring in Hospital	..	1	..	..	1	..	1	1	..	1	..	1	2	..	2	..	..	..	..	..	..	..	6	4	3	1	50	25
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Delirium Tremens—occurring in Hospital	..	..	..	..	..	..	1	..	1	..	4	1	3	..	2	..	..	..	..	..	..	..	11	1	..	..		
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Hæmorrhage after Wounds	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	2	..	2	..		
3. Do. Secondary, after Operations	..	..	..	..	..	..	..	..	2	2	1	..	..	..	..	..	1	..	..	..	..	..	4	2	2	..	50	

TABLE III.

Causes of Death.

No. of Cases	Sex		Age	Disease, Injury, or Operation	Cause of Death, and Date after Admission or Operation
1	...	F.	65—75	Erysipelas	Continuance of Disease. 5th day.
1	M.	...	25—35	Phlegmonous do.	Pyæmia. 22nd day.
3	M.	...	45—55	„ „	Pyæmia. 16th day. Pyæmia. 6th day. Exhaustion. 21st day.
1	M.	...	65—75	„ „	Exhaustion. 32nd day.
1	...	F.	25—35	Diffuse Cellular Inflammation	Bronchitis. 55th day.
1	M.	...	25—35	Acute Pyæmia	Continuance of Disease. 5th day.
1	M.	...	35—45	„ „	„ „ 3rd day,
1	M.	...	15—25	Secondary Syphilis	Tuberculosis. 27th day.
2	M. 1	F. 1	25—35	„ „	(M.) Exhaustion. 12th week. (F.) Exhaustion. 31st day.
1	M.	...	55—65	„ „	Exhaustion. 81st day.
1	...	F.	15—25	Gonorrhœa	Typhoid Fever. 16th day.
1	...	F.	25—35	„	Tuberculosis. 68th day.
3	M. 2	F. 1	45—55	Cancer	(M.) Morb. Cord. 5th day. Asphyxia. 10th day. (F.) Continuance of Disease. 86th day.
1	...	F.	25—35	Tumours	Exhaustion. 16th week.
2	M. 1	F. 1	65—75	Secondary Mortification	(M.) Exhaustion. 7th day. (F.) Exhaustion. 7th day.
1	M.	...	25—35	Scrofula	Tuberculosis. 8th week.
5	M. 4	F. 1	15—25	Psoas Abscess	(M.) Paraplegia. Bedsores. 13th week. Exhaustion. 8th week. Exhaustion. 23rd week. Exhaustion, Vomiting. 47th day. (F.) Exhaustion. 16th week.
2	M.	...	25—35	„	Exhaustion. 17th week and 21st week.
1	M.	...	10—15	Tetanus	Continuance of Disease. 1st day.
1	M.	...	25—35	„	„ „ 1st day.
1	M.	...	45—55	„	„ „ 1st day.
1	M.	...	25—35	Disease of Veins	Rupt. Varicose Vein. Hæmorrhage. 1st day.

TABLE III.—*continued.*

No. of Cases	Sex		Age	Disease, Injury, or Operation	Cause of Death, and Date after Admission or Operation
1	M.	...	55—65	Stricture of Œsophagus	Continuance of Disease. 18th day.
2	M. 1	F. 1	Under 5	Stomatitis	(M.) „ „ 1st day. (F.) do. 5th day.
1	M.	...	35—45	Hernia, Inguinal	Morb. Cord. 4th day.
1	M.	...	65—75	„ „	Peritonitis. 1st day.
1	M.	...	25—35	Stricture of Urethra	Uræmia. 12th day.
2	M.	...	45—55	„ „	„ 3rd day. Albuminuria, Bronchitis. 14th day.
1	M.	...	35—45	Extravasation of Urine	Uræmia. 18th day.
1	M.	...	45—55	„ „	Cystitis, Dis. Kidneys. 7th day.
1	...	F.	15—25	Synovitis	Typhoid Fever. 50th day.
1	...	F.	35—45	„	Pyæmia. 11th week.
2	M. 1	F. 1	5—10	Necrosis	(M.) Pyæmia. 16th day. (F.) Pyæmia. 12th day.
1	M.	...	15—25	„	Sunstroke. 24th day.
1	M.	...	25—35	„	Exhaustion. 10th week.
1	...	F.	5—10	Disease of Joints	Tuberculosis. 28th day.
1	M.	...	10—15	„ „	Exhaustion. 15th week.
2	M. 1	F. 1	15—25	„ „	(M.) Tuberculosis. 12th week. (F.) Tuberculosis. 13th week.
1	M.	...	25—35	„ „	Pyæmia. 5th day.
1	M.	...	15—25	„ Glands	Erysipelas. 9th day.
1	M.	...	10—15	Carbuncle	Exhaustion. 5th day.
1	...	F.	15—25	„	Erysipelas. 3rd day.
1	M.	...	45—55	Abscess	Albuminuria. Bronchitis. 27th day.
1	M.	...	65—75	Chronic Abscess	Exhaustion. 21st day.
1	...	F.	35—45	Ulcer	Pyæmia. 24th day.
1	...	F.	55—65	„	Bronchitis. Anasarca. 10th day.
14	M. 8	F. 6	Under 5	Burn and Scald	(M. 6) Collapse. (M. 2) Exhaustion. (F. 4) Collapse. (F. 1) Convulsions. 15th day. (F. 1) Pneumonia.
3	M. 2	F. 1	5—10	„ „	(M. 2) Collapse. (F. 1) Exhaustion. 30th day.

TABLE III.—*continued.*

No. of Cases	Sex		Age	Disease, Injury, or Operation	Cause of Death, and Date after Admission or Operation
4	...	F.	15—25	Burn and Scald	(3) Collapse. 1st day, 1st day, 4th day. (1) Tetanus. 17th day.
1	...	F.	25—35	„ „	Exhaustion. 16th day.
2	M. 1	F. 1	35—45	„ „	(M.) Collapse. 1st day. (F.) Tetanus. 16th day.
1	...	F.	45—55	„ „	Collapse. 1st day.
1	...	F.	5—10	Contusions	Mortal Injury. 1st day.
2	M.	...	10—15	„	„ „ 1st day, 1st day.
1	M.	...	15—25	„	Pneumonia. 4th day.
3	M. 1	F. 2	25—35	„	(M.) Mortal Injury. 1st day. (F.) Rupture of Abdominal Viscus (p. m.). 1st day, 3rd day.
3	M. 2	F. 1	35—45	„	(M.) Mortal Injury. 4th day. Peritonitis from Rupture of Gall Bladder. 6th day. (F.) Paraplegia. Bedsores. 66th day.
1	M.	...	45—55	„	Chronic Peritonitis. 36th day.
1	...	F.	5—10	Sprains	Meningitis. 61st day.
1	M.	...	10—15	„	Pneumonia. 5th day.
1	M.	...	45—55	Gunshot wounds	„ 23rd day.
1	M.	...	10—15	Laceration of Limbs	Rupture of Abdominal Viscera. 1st day.
2	M. 1	F. 1	35—45	Wounds	(M.) Tetanus. 28th day. (F.) Erysipelas. 10th day.
1	M.	...	55—65	„	Cystitis. 20th day.
2	M. 1	F. 1	65—75	„	(M.) Erysipelas. 36th day. (F.) Ch. Bronchitis. 7th day.
1	M.	...	5—10	Concussion	Meningitis. 3rd day.
1	M.	...	15—25	„	Abscess Brain. 33rd day.
2	M.	...	25—35	Fractures, simple. Base of Skull.	Mortal Injury. 1st day. 5th day.
1	M.	...	35—45	„ „ „	„ 13th day.
2	M. 1	F. 1	55—65	„ „ „	(M.) Mortal Injury. 1st day. (F.) do. 4th day.
1	...	F.	65—75	„ „ „	„ 7th day.

TABLE III.—*continued.*

No. of Cases	Sex		Age	Disease, Injury, or Operation	Cause of Death, and Date after Admission or Operation
1	M.	...	45—55	Fractures, Simple. Cervical Spine	Mortal I jury. 4th day.
1	M.	...	25—35	„ „ Dorsal „	Bedsores. Exhaustion. 47th day.
1	M.	...	45—55	„ „ „ „	„ „ 10th week.
1	M.	...	25—35	„ „ Lumbar „	„ „ 28th day.
1	M.	...	15—25	„ „ Ribs	Injury to Lung. 1st day.
1	M.	...	35—45	„ „ „	Pneumonia. 1st day.
1	M.	...	55—65	„ „ „	„ 1st day.
1	M.	...	75—85	„ „ „	Bronchitis, do. Exhaustion. 7th week.
1	...	F.	65—75	„ „ Femur-cervix	Bedsores. Exhaustion. 40th day.
2	M. 1	F. 1	Over 85	„ „ „	(M.) Jaundice. Exhaustion. 52nd day. (F.) Exhaustion. 29th day.
1	...	F.	10—15	„ „ Tibia and Fibula	Diffuse Cellulitis. Pyæmia. 31st day.
1	M.	...	—5	„ „ compound. Vault of Skull	Mortal Injury. 1st day.
1	M.	...	5—10	„ „ „ „	„ 1st day.
1	M.	...	65—75	„ „ „ „	„ 9th day.
1	M.	...	15—25	Fractures, compound. Femur	Collapse. Extensive other injuries. 1st day.
1	M.	...	45—55	„ „ Tibia and Fibula	Delirium Tremens. Diarrhœa. 36th day.
1	M.	...	65—75	„ „ „ „	Secondary Gangrene. 16th day.
1	M.	...	55—65	Suicide	Diffuse Cellulitis. 10th day.
1	M.	...	65—75	„	Hæmorrhage. 1st day.
1	M.	...	50—60	Primary Amputation. Leg	Exhaustion. 19th day.
*1	M.	...	40—50	Sec. „ Thigh	„ from Hæmorrhage. 4th day.
1	M.	...	40—50	„ „ Foot	Tetanus. 1st day. (Existing prior to operation, and on account of which disease it was performed.)
1	M.	...	10—15	Amputation for Disease. Thigh	Pyæmia. 34th day.
2	M. 1	F. 1	40—50	„ „ „	(M.) Pyæmia. 19th day. (F.) Pyæmia. 15th day.
1	M.	...	50—60	„ „ „	Exhaustion. 16th day.
1	M.	...	50—60	„ „ at Knee-Joint	„ 10th day.

TABLE III.—*continued.*

No. of Cases	Sex		Age	Disease, Injury, or Operation	Cause of Death, and Date after Admission or Operation
1	M.	...	20—30	Amputation for Disease. At Leg . . .	Pyæmia. 9th day.
1	M.	...	15—20	„ „ Ankle-Joint .	„ 40th day.
1	M.	...	40—50	Herniotomy, Inguinal (with opening of sac) .	Peritonitis. (Sloughing of Mesentery, P. M.) 3rd day.
1	M.	...	50—60	„ „ „ „	Peritonitis (existing at time of operation). 1st day.
4	...	F.	50—60	„ Femoral „ „	(3) Gangrene of Gut. 1st day, 3rd day, 6th day. (1) Apoplexy. 56th day.
2	M. 1	F. 1	60—65	„ „	(M.) Gangrene of Gut. 1st day. (F.) Continuance of symptoms. Exhaustion. 29th day.
1	M.	...	65—70	„ „	Peritonitis. Sec. Hæmorrhage. 9th day.
1	...	F.	70—75	„ „	Pyæmia. 10th day.
1	M.	...	40—50	„ „ (without opening of sac)	Peritonitis. 5th day. (No P. M.)
1	M.	...	10—15	„ Umbilical, with „ „ .	Morb. Cord. 1st day.
1	M.	...	65—70	„ „	Gangrene Gut. 3rd day.
1	M.	...	Under 5	Lithotomy	Chronic Peritonitis. 39th day.
1	M.	...	65—70	Lithotrity	Disease of Bladder and Kidneys. 59th day.
1	...	F.	65—70	Excision of Cancer of Breast . . .	Gangrene of Wound. 8th day.
1	M.	...	20—30	„ of Cancerous Tumours . . .	Pyæmia. 10th day.
1	M.	...	30—40	„ „ „ . . .	Recurrence of Disease. 32nd day.
1	...	F.	20—30	„ of Non-Cancerous Tumours (Exostosis)	Pyæmia. 34th day.
1	M.	...	30—40	„ of bones for disease (superior maxilla)	Tuberculosis. 31st day.
1	M.	...	40—50	Trephining Tibia	Exhaustion. 14th week.
1	M.	...	70—75	Removal segment Femur	„ 43rd day.
1	M.	...	15—20	Excision Knee-Joint	Pyæmia. 31st day.
1	M.	...	50—60	Trephining for Injury, Skull . . .	Laceration of Brain. 1st day.
5	M. 2	F. 3	Under 5	Tracheotomy	(M. 2 F. 2) Continuance of Disease. 1st, 2nd, 3rd, and 4th days. (F. 1) Pneumonia. 12th day.
1	...	F.	5—10	„	Continuance of Disease. 1st day.
1	...	F.	40—50	Œsophagotomy	Exhaustion. 18th day.

TABLE III.—*continued.*

No. of Cases	Sex		Age	Disease, Injury, or Operation	Cause of Death, and Date after Admission or Operation
1	...	F.	15—20	Ovariotomy	Hæmorrhage. 1st day.
1	...	F.	20—30	„	Exhaustion. 27th day. (Incomplete operation.)
2	...	F.	30—40	Ovariotomy	Collapse. 1st day, 2nd day.
1	...	F.	40—50	„	Chronic Peritonitis. 36th day.
1	M.	...	40—50	Sections of soft parts (Fistula in Ano) . .	Pyæmia. 33rd day.
1	M.	...	40—50	„ „ (Perineal Section for Extravasation)	Exhaustion from Disease. 1st day.
1	M.	...	Under 5	Reparatory Operations (Artificial Anus) .	„ „ 1st day.
1	...	F.	20—30	„ „ (for Laceration of Perineum) . .	Peritonitis. 5th day.
*1	M.	...	40—50	Ligature Femoral Artery for false Femoral Aneurism	Secondary Hæmorrhage. 15th day.
1	M.	...	40—50	„ Popliteal Aneurism . . .	Phlebitis. Pyæmia. 10th day.
1	M.	...	20—30	„ Femoral Artery for Wound . .	Exhaustion from Secondary Hæmorrhage. 1st day.

* Refer to the same Patient.

TABLE IV.

SHOWING THE WHOLE NUMBER OF SURGICAL OPERATIONS PERFORMED, WITH THE AVERAGE MORTALITY OF EACH OPERATION, FOR THE FIVE YEARS FROM 1863 TO 1867 INCLUSIVE.

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
AMPUTATIONS.													
Primary—													
Hip-Joint	...	...	...	...	...	...	...	...	...	...	...	...	...
Thigh	4	1	1	...	...	50	...	...	...	...	6	2	33
Knee-Joint	...	...	...	1	...	...	...	...	...	...	1	...	...
Leg	4	6	4	4	3	25	66·66	75	25	33·33	21	10	47·61
Foot	3	...	...	...	1	...	...	...	...	...	4	...	...
Toes	2	1	2	2	6	...	...	50	...	...	13	1	7·69
Shoulder-Joint	...	1	...	...	...	...	...	...	...	...	1	...	...
Arm	1	4	1	2	1	...	25	...	...	...	9	1	11·11
Fore-arm	1	3	4	2	1	100	...	...	...	...	11	1	9·09
Hand	...	2	2	2	...	...	...	...	...	...	6	...	...
Fingers	29	18	17	13	7	...	...	...	...	...	84	...	...
Secondary—													
Hip-Joint	...	...	...	...	...	...	...	...	...	...	...	...	...
Thigh	3	3	6	4	2	100	100	50	50	50	18	12	66·66
Knee-Joint	...	...	...	1	...	...	...	...	...	...	1	...	...
Leg	6	1	...	2	1	50	100	50	...	...	10	5	50
Foot	...	...	...	1	1	...	...	...	...	100	2	1	50
Toes	...	...	...	...	1	...	...	...	...	...	1	...	...
Shoulder-Joint	...	...	...	...	...	...	...	...	...	...	...	...	...
Arm	2	6	1	1	...	50	33·33	...	...	...	10	3	30
Fore-arm	3	...	1	...	1	33·33	...	...	...	...	5	1	20
Hand	...	...	...	...	...	...	...	...	...	...	...	...	...
Fingers	...	2	3	...	5	...	...	...	...	...	10	...	...

TABLE IV.—*continued.*

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
AMPUTATIONS—*continued.*													
For Disease—													
Hip-Joint	...	...	...	...	...	...	...	...	...	...	...	...	...
Thigh	22	21	14	5	9	45·45	38·08	50	20	44·44	71	30	42·25
Knee-Joint	...	...	1	...	2	...	...	...	...	50	3	1	33·33
Leg	5	10	14	3	2	...	40	42·85	66	50	34	13	38·23
Ankle-Joint	...	2	...	1	4	...	...	...	...	25	7	1	14·28
Foot	2	...	...	1	...	...	...	...	...	...	3	...	...
Toes	3	1	5	7	1	...	...	...	...	...	17	...	...
Shoulder-Joint	...	...	1	...	...	...	...	...	...	...	1	...	...
Arm	1	1	5	2	3	...	...	...	...	...	12	...	...
Fore-arm	1	3	3	1	1	...	...	...	...	...	9	...	...
Hand	...	...	...	...	...	...	...	...	...	...	...	...	...
Fingers	21	15	8	10	12	...	6·66	12·5	...	...	60	2	3·03
HERNIA.													
Inguinal—													
Radical Cure	...	4	...	...	...	...	25	...	...	...	4	1	25
Herniotomy—Opening Sac	11	9	8	5	3	55·55	88·88	50	60	66·66	36	23	63·88
Without opening	3	4	1	3	...	33·33	...	...	33·33	...	11	2	18·18
Femoral—													
Radical Cure	...	...	...	...	...	...	...	...	...	...	...	...	...
Herniotomy—Opening Sac	12	9	15	10	10	66·66	33·33	33·33	40	80	56	28	50
Without opening	1	2	4	5	5	...	...	25	...	20	17	2	11·76
Umbilical Hernia—													
Opening Sac	4	2	1	...	2	100	100	100	...	100	9	9	100
Without opening	...	...	1	...	...	...	...	...	...	...	1	...	...

TABLE IV.—*continued.*

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
FOR CALCULUS.													
LITHOTOMY	7	9	9	15	8	14·28	11·11	44·44	13·33	12·5	48	9	18·75
LITHOTRITY	3	3	5	...	1	...	66·66	20	...	100	12	4	33·33
URETHROTOMY	2	...	4	2	1	...	...	...	...	...	9	...	...
EXCISION AND REMOVAL OF TUMOURS.													
Cancer of Breast	15	14	16	9	11	13·33	7·14	12·5	...	9·09	55	6	10·90
Other Tumours of Breast	3	7	7	8	9	33·33	...	...	...	...	34	1	2·94
Cancer of Lip	5	5	5	6	3	...	...	20	...	...	24	2	8·33
Exostosis	3	1	3	3	5	33·33	...	...	...	20	15	1	6·66
Epulis	3	4	4	5	3	...	...	...	...	...	19	...	...
Other Tumours—													
Cancerous or Recurrent	16	14	9	13	22	...	...	...	7·69	9·09	74	3	4·05
Not Cancerous	44	37	46	41	48	3·27	...	4·39	4·87	...	216	5	2·31
Polypi—													
Nasal	2	...	...	2	3	...	...	...	...	...	7	...	...
Uterine	...	1	1	...	1	...	...	...	...	...	3	...	...
Others	1	...	...	1	1	...	...	...	...	...	3	...	...
EXCISION (including TREPHINING) OF BONES AND JOINTS.													
For Disease—													
Shoulder-Joint	...	...	1	...	...	...	...	...	...	...	1	...	...
Elbow-Joint	...	3	5	2	2	...	...	...	...	...	12	...	...
Wrist-Joint	...	...	1	...	...	...	...	100	...	...	1	1	100

TABLE IV.—*continued.*

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
EXCISION OF BONES AND JOINTS—*continued.*													
For Disease—													
Hip-Joint	...	1	...	...	...	...	100	...	...	...	1	1	100
Knee-Joint	1	3	8	6	10	...	33·33	25	33·33	...	28	6	21·42
Ankle	...	...	...	...	...	...	...	...	...	...	...	...	...
Portions of Bone	9	1	3	6	4	22·22	...	...	16·16	25	23	4	17·39
For Injury—													
Skull	1	...	...	1	1	100	...	...	100	100	3	3	100
Spine	...	...	1	...	...	...	...	100	...	...	1	1	100
Ununited Fracture, &c.	5	2	1	1	2	20	...	...	...	...	11	1	9·09
Removal of Sequestra—													
From Head and Face	3	4	1	4	7	...	25	...	25	...	19	2	10·52
„ Trunk	...	3	1	1	3	...	...	...	...	...	8	...	...
„ Upper Extremities	1	7	8	7	10	...	...	...	...	...	33	...	...
„ Lower Extremities	18	20	18	27	33	...	...	...	3·70	9·09	166	4	3·44
TRACHEOTOMY	5	11	1	9	8	80	63·63	...	33·33	75	34	20	58·82
LARYNGOTOMY	...	...	...	...	...	...	...	...	...	...	...	...	...
ŒSOPHAGOTOMY	...	...	...	...	1	...	...	...	...	100	1	1	100
ABDOMINAL SECTION:—													
Ovariotomy	1	...	2	9	6	100	...	100	44·44	83·33	18	12	66·66
Gastrotomy	...	...	...	...	...	...	...	...	...	...	...	...	...
Opening of Colon	...	...	...	1	...	...	...	...	...	...	1	...	...
Cæsarean Section	1	...	...	1	...	100	...	...	100	...	2	2	100

TABLE IV.—*continued.*

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
REPARATORY OPERATIONS.													
Cicatrices and Contractions (from Burns and other Injuries)	3	1	...	1	2	...	...	...	...	...	7	...	...
Deformities from Disease	1	4	4	3	...	100	...	...	...	...	12	1	8·33
" " Injuries	2	8	6	12	14	...	25	...	8·33	7·14	42	4	9·52
Deformities, Congenital—Hare Lip	9	5	2	5	2	11·11	...	...	...	...	23	1	4·34
Others	...	1	2	1	1	...	...	...	...	...	5	1	20
Cleft or Defective Palate	2	2	1	1	6	...	...	...	...	...	12	...	...
PARACENTESIS.													
Hydrocele and Hæmatocele	11	16	14	8	22	...	6·25	...	...	...	71	1	1·40
Ovarian	...	1	1	...	...	...	100	...	...	...	2	1	50
Thoracic	1	2	...	...	1	100	...	...	...	...	4	1	25
Vesical	3	3	1	2	1	...	100	...	...	...	10	3	30
Cephalic	...	...	...	...	...	...	...	...	...	...	...	...	...
Spina Bifida	2	...	...	...	...	...	...	...	...	...	2	...	...
EVACUATION OF CYSTS AND TUMOURS.	...	1	...	2	2	...	...	...	...	...	5	...	...
SECTIONS OF SOFT PARTS.													
Perineal Section	5	9	3	2	10	...	22·22	33·33	...	10	29	7	24·13
Fistula	9	6	9	18	21	...	...	...	5·55	4·76	63	2	3·17
Anal and Rectal Ulcers	...	...	...	1	...	...	...	...	...	...	1	...	...
Hæmorrhoids	2	...	...	...	...	...	...	...	...	...	2	...	...
Circumcision	8	3	2	11	6	...	...	...	...	...	30	...	...

TABLE IV.—*continued.*

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
SECTION OF SOFT PARTS—*continued.*													
Partial Excision of Organs	...	...	1	3	2	...	...	...	...	...	6	...	...
Others	...	2	...	...	...	...	...	...	...	...	2	...	...
Extraction of Loose Cartilages	...	...	1	...	3	...	...	...	...	...	4	...	...
„ Balls and other Foreign bodies	3	...	1	1	...	...	...	...	...	...	5	...	...
Internal Division of Stricture—													
Urethra	3	...	...	...	...	...	...	...	...	...	3	...	...
Rectum	1	1	...	1	1	...	...	...	...	...	4	...	...
Others	1	...	...	...	...	...	...	...	...	...	1	...	...
TENOTOMY	22	15	24	19	25	...	...	...	...	...	105	...	...
MYOTOMY	...	1	...	...	...	...	...	...	...	...	1	...	...
NEUROTOMY	1	1	...	...	2	...	...	...	...	...	4	...	...
REMOVAL OF PARTS BY LIGATURE.													
Nævi	11	2	3	3	3	...	...	...	...	...	22	...	...
Hæmorrhoids	...	...	3	6	7	...	...	...	...	...	16	...	...
Tongue	...	...	...	...	...	...	...	...	...	...	...	...	...
Others	...	...	...	...	...	...	...	...	...	...	...	...	...
REMOVAL OF PARTS BY ÉCRASEUR	4	2	...	5	2	...	...	...	20	...	13	1	7·69
LIGATURE OF ARTERIES.													
For Aneurism	...	6	1	7	3	...	50	100	...	66·66	17	6	35·22
For Hæmorrhage	14	16	12	12	7	7·14	18·75	...	8·33	14·28	61	6	1·83
For other Diseases and Injuries	...	...	1	1	...	...	...	100	100	...	2	2	100

TABLE IV.—*continued.*

OPERATIONS	Cases under Treatment					Per-centage of Deaths					Total Number of		Average Per-centage of Deaths
	1863	1864	1865	1866	1867	1863	1864	1865	1866	1867	Cases	Deaths	
OBLITERATION OF VEINS . . .	4	2	4	2	3	...	...	...	...	...	15	...	...
OPHTHALMIC OPERATIONS.													
Extraction of Lens	7	3	...	1	7	...	...	...	...	...	18	...	...
Dislocation	1	1	...	...	1	...	...	...	...	...	3	...	...
Keratonyxis	1	...	3	2	...	...	...	...	...	...	6	...	...
Artificial Pupil	1	2	...	...	...	...	...	...	...	...	3	...	...
Iridectomy	...	...	...	...	4	...	...	...	...	...	4	...	...
Strabismus	...	4	2	2	8	...	25	...	...	...	16	1	6·25
Excision of Eye	...	3	6	4	3	...	...	...	...	...	16	...	...
Abcision of Eye	...	...	5	3	...	...	...	...	...	...	8	...	...
Entropion	...	5	...	...	...	...	...	...	...	...	5	...	...
Ectropion	...	...	...	...	1	...	...	...	...	...	1	...	...
Other Operations	1	...	13	...	...	...	...	...	...	...	14	...	...
REDUCTION OF DISLOCATIONS (OLD) .	2	2	2	4	2	...	...	50	...	...	12	1	8·33
RE-FRACTURE OF BONES . . .	1	...	...	...	1	...	...	...	...	...	2	...	...
TOTAL	429	411	410	411	470	14·91	16·54	12·68	9·49	11·49	2131	277	12·99

The preceding tables have been selected for republication from the annual report of the Registrars to the Hospital (Dr. Southey and Mr. Willett), printed by order of the Governors.

ST. BARTHOLOMEW'S HOSPITAL & COLLEGE.

PRESENT MEMBERS.

Consulting Physician, DR. BURROWS.

Physicians to the Hospital.

DR. FARRE, DR. BLACK, DR. EDWARDS, DR. HARRIS.

Consulting Surgeons, MR. SKEY, MR. WORMALD.

Surgeons to the Hospital.

MR. PAGET, MR. COOTE, MR. HOLDEN, MR. SAVORY.

Assistant Physicians to the Hospital.

DR. ANDREW, DR. SOUTHEY, DR. CHURCH, DR. GEE.

Assistant Surgeons to the Hospital.

MR. CALLENDER, MR. T. SMITH, MR. WILLETT, MR. LANGTON.

Physician Accoucheur, DR. GREENHALGH.

SCHOLARS.

1861	Baker, W. Morrant	Newman, William	1854
1856	Barford, James G.	Paine, William	1860
1864	Bateman, F.	Rhind, Samuel	1850
1855	Best, Henry D.	Richards, F. W.	1863
1860	Brook, Charles	Rouch, J. R.	1867
1851	Brown, John A.	Sadler, Michael J.	1855
1866	Butcher, W. D.	Senior, Charles	1858
1852	Callender, George W.	Sharpin, Henry W.	1849
1864	Cole, T.	Shepard, W. L.	1865
1857	Crowfoot, William M.	Smith, Herbert A.	1868
1868	Field, A. F.	Snook, J. W.	1864
1865	Garrett, W. J.	Square, W.	1866
1856	Goodall, Ralph	Thompson, Charles R.	1850
1858	Helm, George F.	Turner, William	1853
1862	Hingston, Albert	Vernon, Bowater J.	1862
1853	Humphry, Frederick A.	Winkfield, William B.	1857
1859	Jeaffreson, Horace		

Abercrombie, J., MD Cantab. 1848, FRCPL 1849
Ackland, W., LSA 1823
Ackland, W. H., MRCS 1847, MD St. And. 1862
Acton, W., MRCS 1840
Adam, J., MRCS 1835, MD Edin. 1855
Adams, E. B., MRCS 1850, LSA 1852
Adams, J., MRCS 1859, MD St. And. 1859
Adams, J., MRCS 1848, LFPS Glas. 1859
Adams, J. D., MRCS 1858, MD St. And. 1862
Adams, J. O., MRCS, LSA 1865
Adams, R., MRCS 1854, LRCP Edin. 1860
Addenbroke, E. H., MRCS 1866
Addison, C. E., MRCS 1862, LKQCP Ireland 1865
Aldrich, P., LSA 1835, MRCS 1859
Alger, J. S., LSA 1831, MRCS 1842
Allas, L., MRCS 1857
Allen, A. B. de Lisle, MRCS 1845
Allen, E., MRCS, LSA 1842
Allen, G., LSA 1845, MD St. And. 1856
Allen, J., MRCS 1855, LSA 1856
Allen, J. W., MRCS 1855, LSA 1857
Allen, R., LSA 1833, MRCS 1834
Alsop, E., LSA 1843, MRCS 1847
Amsden, J. G., LSA 1837, MD Jena 1853
Anderson, D. H. B., MRCS 1866
Anderton, C., MRCS, LSA 1834
Andrew, J., MD Oxon. 1863, FRCP 1866
Andrew, J. M., MRCS 1855
Andrew, W. W., MRCS 1860, MB Cantab. 1861
Andrews, E., MRCS, LSA 1848
Andrews, F. F., MRCS 1850, MD St. And. 1862
Angier, G. A., MRCS 1861, MD St. And. 1862
Angier, J. H., MRCS, LSA 1826
Angove, E. S., MRCS 1867
Antrobus, D., LSA 1836, MRCS 1859
Archer, E., MRCS 1848, MD Paris 1865
Archer, J., LSA 1830, FRCS 1859
Archer, L. H., MRCS 1851
Arding, W., MD Edin. 1827
Armstrong, J., MRCS 1828, MD Aber. 1851
Armstrong, S., MRCS, LSA 1861
Arnold, J., MRCS 1861, LRCP 1866
Arnold, J., MRCS 1867
Ascroft, E. H., MRCS, LSA 1830
Ashford, J. B., MRCS 1843, MD Aber. 1851
Ashton, J. H., MRCS 1863
Atherley, J., in practice prior to 1815
Atkinson, R., LSA 1822, MRCS 1823
Attfield, G., MRCS 1850
Atwood, W. A., MRCS 1860
Austin, T. J., LSA 1842, MRCS 1843

Badger, S., MRCS, LSA 1844
Badley, J. P., MRCS, LSA 1847
Badley, J., MRCS 1803, FRCS 1843
Bage, W. F. J., LSA 1828, MRCS 1830
Bagg, G. W., MRCS 1843, LSA 1845
Bailey, F. C., LSA 1860, MRCS 1864
Bailey, J., LSA, MRCS 1833
Baillie, A., MRCS 1825
Baillie, M. T. B., LSA 1849, MRCS 1858
Bainbridge, W., MRCS, LSA 1833
Baker, A., MD Montreal 1848
Baker, A., MRCS 1837, FRCS 1852
Baker, J., LSA 1828, LRCPE 1859
Baker, R., LSA 1820, MRCS 1821
Baker, R. L., LSA 1841, FRCS 1861
Baker, S. I., MRCS, LSA 1853
Baker, T. J., LSA, MRCS 1833
Baker, W., MRCS 1811
Baker, W. M., MRCS 1861, FRCS 1864
Ball, D., LSA 1824, FRCS 1844
Baller, J. H., MRCS 1829, MD St. And. 1861
Baly, G., MRCS 1855
Bancks, T., LSA 1834, FRCS 1860
Banks, W. R., LSA 1853, MD St. And. 1855
Barbeau, A., MRCS 1851
Barford, J. G., MRCS 1857, LRCP 1861
Barker, E., LSA 1823, FRCS 1849
Barker, E., jun., MRCS 1856, LSA 1858
Barnes, C. H., LSA 1823, FRCS 1852
Barnes, F. W., MRCS 1845
Barnett, H., LSA 1828, MRCS 1834
Barreau, F. H., MRCS 1859
Barrett, C. A., MRCS 1854, LSA 1859
Barringer, T. S., MRCS 1839, LRCP Edin. 1860
Barrow, B., MRCS 1836, FRCS 1862
Barsham, J. B., MRCS, LSA 1836
Barter, C. S., LSA 1859, FRCS 1865, MB Lond. 1866
Barter, T., LSA 1825, MRCS 1826
Bartlam, E. G., MRCS 1839, LSA 1840
Bartleet, E., LSA 1824, FRCS 1852
Bartley, R. T. H., LSA 1840, MD St. And. 1845
Barton, S., MRCS 1811, FRCS 1844
Barwise, J., LSA 1840 MD St. And. 1862
Basley, C., MRCS, LSA 1840
Bass, A., MRCS 1852, MD St. And. 1857
Bateman, H., LSA 1828, FRCS 1855
Bateman, F., MRCS, LSA 1866, MB Lond. 1866
Batt, A., MRCS 1852, MD St. And. 1857
Batt, C. D., MRCS, LSA 1867
Batten, R. W., MRCS 1857, MD Lond. 1860
Batten, W., LSA 1832
Batty, B., MRCS 1815
Battye, W., MRCS 1864
Baxter, H. F., MRCS 1838
Bazeley, W., MRCS 1861, LSA 1862
Beach, H., LSA 1828, MRCS 1829

Beale, J. S., MRCS 1846, LM 1853
Beale, T. M., LSA 1855, MRCS 1856
Beard, C. T., MB Cantab. 1855, MRCP 1859
Beattie, H. M., MRCS 1861, MD St. And. 1862
Beckett, F. M., MRCS 1864
Beckingsale, J. E., MRCS 1833, LSA 1832, FRCS 1859
Bedwell, H., MRCS 1840, FRCS 1860
Bedwell, J. R., LSA 1831, MRCS 1832
Beecroft, J., MRCS, LSA 1847
Bell, G. W., MD Edin. 1835
Bell, J., LSA 1825
Bendall, J., MRCS 1861, LSA 1862
Benfield, T. W., MRCS 1843, FRCS 1859
Bennett, J. H., FRSE, MD Edin.
Bennett, F. G., MRCS, LSA 1864
Bennett, F., MRCS 1854
Bennett, G. J., MRCS 1859, LSA 1860
Bennett, J, MRCS, LSA 1839
Bennett, C. J., MRCS, LSA 1868
Berridge, E., MB Lond. 1867
Berry, S., LSA 1830, FRCS 1852
Best, F. A., MRCS 1868, LSA 1864
Best, H. D., MRCS, LSA 1855
Betham, J., MRCS 1855
Bett, R. L., MRCS 1859
Beveridge, J. E., MRCS 1836
Bewley, C., MRCS 1852, LSA 1856
Bickersteth, E. R., MRCS 1851, FRCS Edin. 1855
Bicknell, H., MRCS 1855
Bidden, R. C., MRCS 1831
Biddle, H, LSA 1822, MRCS 1823
Billett, J., in practice prior to 1815
Birch, W., MRCS 1823, FRCS 1844
Bird, H., LSA 1834, FRCS 1858, Ext. LRCP Lond. 1858
Bird, J., LSA 1821, MRCS 1825
Bird, J., MRCS, LSA 1834
Birt, A. W., MRCS 1862
Bishop, E., MD. Erlang. 1856
Black, P., MD Oxon. 1839, FRCP 1845
Blackman, F., MRCS 1854, LSA 1855
Blackman, F., MRCS 1856, LM 1858
Blackmore, H. P., MRCS 1857, MD St. And. 1857
Blackmore, S., LSA 1828, MD St. And. 1852
Blaker, E. S., MRCS 1859, LRCP Edin. 1860
Blaker, H. M., MRCS 1839, LSA 1840
Blasson, T., LSA 1823, FRCS 1853
Blenkinsop, H. LSA 1834, FRCS 1852
Bletchly, W. E., MRCS, LSA 1862
Blomfield, J., MD Aber. 1855, FRCS 1855
Bloxam, J A., MRCS 1864, LSA 1866
Blue, W. A. S, MRCS 1868, LRCP Edin.
Blundell, J. W. F, MD Giessen 1851, LRCP Edin. 1859
Blundell, T. L., MRCS 1812, MD Glasg. 1826, FRCS 1852
Blunt, T., MRCS 1863, MD Edin. 1865
Blyth, E. J., MRCS, LSA 1850
Bodilly, J. D., MRCS 1857, LSA 1860
Bodington, G., LSA 1825, LRCP Edin. 1859
Bodman, J. B., LSA 1834
Bogg, E. B., MRCS, MD St. And. 1859
Bogg, J., LSA 1821, MRCS 1822
Bond, E. T., MRCS, LSA 1820
Bond, F. St. Q., MRCS, LSA 1858
Bond, H. J. H., MD Cantab. 1831, FRCP 1835
Bond, W. Y., LSA 1817
Boodle, R. H., MRCS, LSA 1843
Bore, G. H., LSA 1834, MRCS 1835
Borrett, J., MRCS 1827, MD Edin. 1830
Bossy, A. H., LSA 1851, MRCS 1859
Bostock, A. S., MRCS 1867, LSA 1868
Bostock, E. J., MRCS 1864, LSA 1865
Bostock, J. A., MRCS 1838
Bott, T., LSA 1829, *MRCS 1830*
Boughton, J. H., *MRCS 1848*
Boulton, D. F., *MRCS, LSA 1866*
Bowen, W., MRCS 1855
Bowes, R., MRCS, LSA 1831
Box, J., LSA 1826, MRCS 1827
Bradford, R., MRCS 1868
Bradshaw, A. F., MRCS 1857
Brake, W. N., MRCS 1844
Bramley, L., LSA 1829, FRCS 1853
Bratt, W., MRCS, LSA 1849
Brendon, P., MRCS 1817, FRCS 1852
Brent, R., MRCS 1844, MD St. And. 1845
Brereton, C. Le Gay, MRCS 1851
Brewer, A., MRCS 1832, LSA 1834
Brewer, A. H., MRCS 1864, LRCP 1865
Brewer, C. C., MRCS 1864, LSA 1865
Brewer, H. M., MRCS 1864, LSA 1865
Brewer, J., LSA 1825, FRCS 1852
Brewer, T., MRCS, LSA 1863
Brewer, W. H., MRCS 1839
Brickwell, C. J, LSA 1832, MRCS 1833
Brickwell, H., MRCS 1839
Brickwell, J., LSA 1829, MRCS 1830
Brickwell, J., senr., MRCS, LSA 1829
Brickwell, J., MRCS, LSA 1856
Bridger, J., LSA 1857, MRCS 1859
Brigstocke, H., MRCS 1828 MD St. And. 1861
Brigstocke, R. W., MRCS, LSA 1859
Brine, J. E., MRCS 1844, LSA 1845
Bringloe, C., MRCS 1851, LSA 1852
Briscoe, J., MRCS 1842, LSA 1844
Broadbent, J. C., MRCS 1862, LKQCP Ire. 1864
Brodhurst, B. E., MRCS 1844, FRCS 1859
Brodribb, W. P., MRCS 1823, LSA 1822
Bromley, F., MRCS 1853, LSA 1854
Brook, C., MRCS, LSA 1861
Brooke, C, FRS, MA Cantab. 1827, FRCS 1844
Brookman, H., MRCS 1820, LRCP 1842
Broughton, R. D., MRCS, LSA 1865
Brown, F. W., MRCS 1861
Brown, J. A., MRCS, LSA 1851
Brown, J. M., MRCS 1851
Brown, J. W., MRCS, LSA 1861
Brown, R. G., MRCS, LSA 1854
Brown, N. B., LSA 1829, MRCS 1830
Browne, B., LSA 1832, LRCP Edin. 1859
Browne, F. H., MRCS, LSA 1860
Brownless, A. C., MRCS 1841

Bruce, J., MRCS 1863
Brunskill, W., LSA 1823
Brush, J. R., MD Heid. 1839, FRCS, MRCP 1860
Bryan, E., LSA 1863, MRCS 1864
Bryan, J. M., MRCS 1833, MD Aber. 1852
Bryant, W. H., MRCS 1864
Buchanan, W., LSA 1814, MD Erlang. 1841
Buck, H. J., MRCS 1859, LRCP Edin. 1860
Buckle, F., MD St. And. 1862, LRCP 1863
Buckley, N., FLS, MRCS 1842, MD St. And. 1843
Bucknill, H. W., MRCS, LSA 1851
Bullen, G., senr., MRCS 1813, FRCS 1844
Bullmore, F. C., LSA 1830, MRCS 1831
Bullocke, J. J. A., LSA 1824, MD New York 1825
Bulmer, W., LSA 1828, MRCS 1829
Bunn, W. P., MRCS, LSA 1819
Burd, E., MB 1851, MD Cantab. 1859
Burgess, J. MRCS 1843, MD Aber. 1847
Burlton, T., LSA 1834, FRCS 1854
Burman, T. S., LSA 1836, MRCS 1838
Burn, W. B., MRCS 1865
Burroughs, J. B., LSA 1827, MRCS 1828
Burrows, G., FRS, MD Cantab. 1831, FRCP 1832
Burrows, W. A., LSA 1852, MRCS 1859
Burt, G. R., LSA 1835, LRCP Edin. 1860
Burt, S. J., MRCS 1830, LSA 1853
Burton, B. T, MRCS 1839, LSA 1840
Bury, J. W., MRCS 1858, MD Giessen 1865
Bush, E., LSA 1828, MRCS 1829
Bush, J., LSA 1867
Bushnan, J. S., LRCS 1830, MD Heid. 1836
Buswell, R., LSA 1853, MRCS 1854
Butler, W. H., MRCS 1829
Butler, T. M., MRCS, LSA 1860
Butlin, C. H., MRCS 1863
Butlin, H. T., MRCS 1867
Button, B., MRCS, LSA 1847

Caird, T. W., MRCS 1850, LSA 1851
Caird, W., LSA 1825, MRCS 1826
Callaway, E., MRCS 1858, LSA 1861
Callaway, Rev. H., MRCS 1842, MD Aber. 1853
Callender, G. W., MRCS 1852, FRCS 1855
Cam, T., MRCS 1837, FRCS 1844
Camden, G. J., LSA 1825, MRCS 1826
Campion, H., MRCS 1856
Candy, J., LSA 1827, MD Aber. 1860
Cann, T., MRCS 1859, MD St. And. 1860
Cape, L., MD Edin. 1833, FRCP Lond. 1857
Carden, H. D., MRCS 1829, FRCS 1843
Carter, A. P., MRCS 1858, LSA 1859
Carter, D. D., LSA 1830, MRCS 1831
Carter, J., LSA 1836
Cartwright, J. T., MRCS 1832, LSA 1834
Cary, W. H., LSA 1823, MRCS 1825
Cattell, T. W., MRCS, LSA 1831
Cattlin, W. junr., MRCS 1859, LDS 1860
Cautley, J., MRCS 1852, MD St. And. 1852
Cautley, W. C., MRCS, LSA 1849
Chadwick, C., MD Edin. 1837, MRCP Lond. 1859
Chadwick, P. C., MRCS, LSA 1835
Chamberlaine, J., LSA 1852, MRCS 1860
Chance, F., MB Camb. 1855, FRCP Lond. 1863
Chapman, E. G., MRCS 1848, LSA 1849
Chapman, G., LSA 1822, MRCS 1823
Chapman, H. T., MRCS 1828, FRCS 1843
Chapman, T., LSA 1829, MRCS 1840
Chappell, J. J., MRCS 1857, MD St. And. 1859
Charlton, E., MD Edin. 1836, MD Durham 1856
Chatto, J., MRCS, LSA 1832
Chavasse, T., LSA 1822, FRCS 1844
Cheese, J., MRCS 1860
Chippendale, W., MRCS 1855, MD St. And. 1862
Cholmeley, W., MD St. And. 1850, MRCP 1859
Cholmondeley, J., MRCS 1816
Chune, H. C., MRCS 1860
Church, W. J., LSA 1821, FRCS 1852
Church, W. S., MB Oxon. 1864, MRCP 1864
Churchill, S., MRCS 1848, MD Aber. 1849
Clapham, L., MRCS 1867
Clark, A., MRCS 1846, LSA 1850
Clark, J. E., LSA 1822, MRCS 1830
Clark, J. L., LSA 1835, MRCS 1854
Clark, R. O., MRCS, LSA 1848
Clarke, E. G., MRCS 1861
Clarke, J. S., LSA 1833, MD King's Coll. Aber. 1861
Clarke, W. M., MRCS, LSA 1850
Clifton, N. H., LSA 1841, FRCS 1856
Close, H. A., MRCS 1859, LRCP 1863
Clubbe, W. H., MRCS, LSA 1850
Coalbank, T., MRCS, LSA 1866
Coates, G., LSA 1827
Coates, G. V., LSA 1852, MRCS 1854
Coates, M. G. W., LSA 1836, MRCS 1838
Coates, W. M., LSA 1832, MRCS 1833
Coats, G. A. A., MRCS 1864
Coathupe, C., MRCS 1862
Cocker, J., LSA 1827, MD Erlang. 1845, FRCP Edin. 1858
Cocker, W. H., MRCS 1861, LSA 1862
Cockey, E., MRCS, LSA 1842
Cole, T., MRCS 1866, MB Lond. 1866
Coleman, A., MRCS, LDS 1860
Coleridge, E., MRCS 1826
Collins, C. H., LSA 1845, MRCS 1846
Collins, J. B., MRCS, LSA 1860
Collyns, G. N., MRCS, LSA 1855
Collyns, J. B., MRCS 1845, LSA 1846
Collyns, W., LSA 1844, MRCS 1845
Colquhoun, A. G., MRCS 1861
Colthurst, J., LSA 1831, FRCS 1844
Combe, H., MRCS 1808
Compton, T. A., MRCS 1864, LRCP 1865
Cooke, A., LSA 1832, MD Albany 1833
Cooke, A. S., LSA 1863, MRCS 1864
Cooke, W., MRCP 1866, MD St. And. 1822
Cooke, W., MRCS 1822, LM 1860
Cooke, W. R., MRCS 1838, LSA 1839

Cooke, W. H., MRCS 1855, MD Aber. 1860
Coombs, J., MRCS 1859, MD Erlang. 1864
Coombs, R H., LSA 1866, MRCS 1867
Cooper, A. MRCS 1861 FRCS 1868
Cooper, Sir H., MRCS 1830, MD Lond. 1841
Cooper, F. S., LSA 1839, FRCS 1854
Cooper, W. W., MRCS 1838, FRCS 1846
Coote, H., MRCS 1838, FRCS 1844
Cope, R., MRCS 1857
Copeland, G. F., MRCS 1832
Corfe, G., LSA 1831, MD Aber. 1849
Cornish, S. H., MRCS 1854, LSA 1856
Cornwall, J., MRCS 1840, LSA 1841
Corsellis, N. C., LSA 1820, MRCS 1831
Costall, J., MRCS 1816
Costelloe, D., MRCS 1867
Costerton, H., MRCS 1852, LSA 1857
Cotterell, T. R., MRCS 1837, LSA 1845
Cotterell, T. S., MRCS 1833, LSA 1834
Cotton, T., MRCS 1860, MD St. And. 1861
Courtney, C. F. A., MRCS, LSA 1847
Coveney, J. H., MRCS, LSA 1843
Covey, C. E., MRCS 1863, LSA 1865
Covey, G., MRCS 1830
Covey, G., jun., MRCS 1864, LSA 1865
Covey, W. H., MRCS 1826, FRCS 1852
Cowen, H. L., MRCS 1839, FRCS 1859
Craven, R. M., MRCS 1846, LSA 1847
Crawford, D., MRCS 1820
Cregeen, J. J., LSA 1851, MD St. And. 1853
Cresswell, J., MRCS, LSA 1841
Cripps, E., MRCS 1845, LSA 1846
Croft, J. Mc G., MRCS 1846, MD Aber. 1856
Croft, R.C., MRCS 1853, LRCP Edin. 1859
Crompton, S., MRCS 1833, MD St. And. 1862
Crookes, J. F., MRCS 1833, FRCS 1843
Cropp, F. J., MRCS 1864, LRCP Edin. 1864
Crosby, T., MRCS 1831, LSA 1832
Cross, W., LSA 1837, FRCS 1852
Crosse, T. W., LSA 1847, FRCS 1860
Crowfoot, E. B., MRCS, LSA 1867
Crowfoot, W. E., LSA 1828, FRCS 1845
Crowfoot, W. J., MRCS 1813, MD Leyden 1830
Crowfoot, W. M., MRCS 1858, MB Lond. 1859
Cuddeford, T., MRCS, LSA 1865
Cuffe, R., LSA 1852, MRCS 1856
Culling, R., MRCS 1843, LSA 1860
Cupiss, F. P., LSA 1850, MRCS 1851
Curgenven, J. B., MRCS, LSA 1852
Curling, T. B., FRS, FRCS 1843
Currie, J. L., MRCS 1863, LRCP Lond. 1864
Currie, J. T., LFPS Glas. 1838
Curtis, A., MRCS 1857, LSA 1860
Curtis, C. E., MRCS 1865
Curtis, F., MRCS, LSA 1822
Curtis, W., LSA 1824, MRCS 1825
Cutcliffe, J. L., MRCS, LSA 1847

Daglish, G., LSA 1826, FRCS 1853
Dain, F. R., MRCS 1839, LSA 1843
Dalley, C. T., MRCS 1864, LSA 1865
Dakeyne, B. B., MRCS 1833, LSA 1834
Dalley, W. C., LSA 1823
Dalley, W. C., jun., MRCS, LSA 1855
Daly, O., FRCSI 1844, MD Dublin 1857
Danford, F., MRCS, LSA 1838
Daniel, J. S., LSA 1825, FRCS 1852
Daniel, T. P., MRCS 1858, LSA 1859
Daniel, W. J., MRCS, LSA 1856
Daniel, W., LSA 1861, MRCS 1862
Daniell, A. H., MRCS 1848, LSA 1850
Daniell, W., LSA 1838, MRCS 1840
Daniell, W. A., MRCS 1866
Daniell, W. C., MRCS 1856, LSA 1857
Dansey, G. F., MRCS 1861
Dashwood, W. H., MRCS 1858
Davenport, C., MRCS 1852, LRCP Edin. 1860
Davey, A. G., MRCS 1860, MD St. And. 1862
Davey, J. G., LSA 1833, MD St. And. 1863
Davey, R. G., LSA 1830, MRCS 1858
Davey, R. S., LSA 1853, MD St. And. 1862
Davies, F., LSA 1834, FRCS 1852, MD St. And. 1862
Davies, H., MRCS, LSA 1860
Davies, H. H., MRCS, LSA 1858
Davies, J., MRCS 1819, MD Aber. 1850
Davies, J., MD Aber. 1845
Davies, N. E., MRCS, LSA 1866
Davies, T., LSA 1824, MRCS 1827
Davies, T., MRCS 1864
Davies, T. G. D., LSA 1852, MRCS 1853
Davies, T. H. W., MRCS 1864, LSA 1865
Davies, W., MRCS, LSA 1847
Davis, A. A., MRCS 1854, LRCP Edin. 1859
Davis, E., in practice prior to 1815
Davis, T., MRCS, LSA 1823
Davis, T., jun., MRCS 1855, MD Lond. 1861
Davy, J. W., LSA 1829
Dawson, J., LSA 1831, MRCS 1860
Dawson, R. H., MRCS, LSA 1861
Dawson, T., MRCS, LSA 1835
Dawson, W., LSA 1834, MRCS 1835
Dawson, W. H., MRCS, LSA 1860
Day, E. H., MB Cantab. 1856, MRCS 1862
Day, J. S., LSA 1841
Dayman, H., LSA 1832, FRCS 1854
Deane, J., MRCS 1834, MD Glas. 1854
Deane, R., LSA 1828
Dearden, J., MRCS, LSA 1856
Dearden, J., MRCS, LSA 1858
De Crespigney, E. C., MRCS 1845
Delagarde, J. L., MRCS 1855, MB Lond. 1857
Delagarde, P. C., MRCS 1819, FRCS 1843
Denne, T., MRCS 1857
Denny, C. J., LM Dub, MRCS 1864
Denny, J., MRCS 1857, LRCP Edin. 1860
Dew, E., MD Edin. 1831, MRCP 1831
Dewes, E., MRCS 1843, MD Glas. 1844
Diamond, H. W., LSA 1829, MD Kiel 1834
Dickin, J., FRCS 1843
Dickinson, T., LSA 1819
Dingley, W., MRCS, LSA 1850
Diver, T., LSA 1857, MD St. And. 1858
Dixon, C., LSA 1816, MRCS 1819
Dixon, T. G., MRCS 1841, MD St. And. 1853
Dobell, H. B., MRCS 1849, MD St. And. MRCP 1856

Dolton, W. B, MD Heild. MRCP 1859
Donald, J. R., LSA 1838, FRCS 1865
Douglas, R., LSA 1823, MRCS 1825
Dover, F., MRCS 1829, FRCS 1843
Dow, J., MRCS 1858, LSA 1857
Dowker, F. W., MRCS, LSA 1859
Dowling, T., LSA 1823, MRCS 1860
Drage, C., MRCS 1848, MD Aberd. 1857
Drake, A., LM Dub. 1848, MB Cantab. 1849
Druce, W., LSA 1857
Ducket, C. A., MRCS 1852, MD St. And. 1862
Duckworth, D, MD Edin. 1863, MRCP Lond. 1865
Dudley, J. G., MRCP 1859, MD Cantab. 1860
Dudley, W. L., MRCS 1846, MD St. And. FRCS 1860
Duff, W. H., MRCS, LSA 1838
Dunderdale, W., MRCS 1862, MD St. And. 1862
Dunn, A., MRCS 1862
Dunn, C. B. N., MRCS 1860
Dunn, G, MRCS, LSA 1848
Dunne, F. W., MRCS 1855, LSA 1856

Eaton, F. J., MRCS 1855, LSA 1858
Eaton, J. C, MRCS, LSA 1866
Earle, E. S., MRCS 1856, FRCS 1860
Ebbage, LSA 1833, FRCS 1858
Eccles, A., MRCS 1843, FRCS 1858
Eccles, G. H., MRCS 1862, LRCP Edin. 1865
Eccles, J. H., MRCS, LSA 1839
Eccles, W. S, MRCS 1864, LSA 1865
Eck, V. F., MRCS 1864
Edginton, G. W, LSA 1818
Edis, W., MB Lond. 1861
Edis, A. W., MB Lond. 1863
Edkins, C., LSA 1832, MRCS 1833
Edlin, E. H., MRCS, LSA 1862
Edmunds, J, MRCS 1852, LSA 1852
Edwardes, C. L., MRCS 1856, LSA 1858
Edwardes, T., MRCS 1835, LSA 1837
Edwards, C. T., MRCS 1833
Edwards, H. N., MRCS 1864, LSA 1865
Edwards, D. P., MRCS 1828, FRCS 1860
Edwards, G. N., MD Cantab. 1859, FRCP 1863
Edwards, H. J., MRCS, LSA 1857
Edwards, W., MRCS, LSA 1861
Edye, S., MRCS 1859, LSA 1860
Egles, G. M, MRCS, LSA 1860
Eland, J. L., MRCS 1836
Eldershaw, F, MRCS 1860
Elkington, T., MRCS 1854, LRCP Edin. 1860
Elliot, R. L., MRCS 1862, LRCP 1863
Elliott, J., LSA 1826, MRCS 1827
Elliott, G, MRCS 1862
Elliott, W., LRCS Edin. 1815, MD 1828
Elliott, E., MRCS 1839, MD Aberd. 1852
Elliott, G. H., MRCS, LSA 1863
Elliott, J. R., MRCS 1862, LRQCPI 1863
Elliott, P. MRCS 1834
Ellis, H. D., MRCS 1861, LSA 1863
Ellis, T. S., MRCS, LSA 1861
Ellis, W. H., MRCS, LSA 1866

Ellison, J., MRCS 1840, MD Lond. 1845
Else, J. O., MRCS, LSA 1825
English, T., MRCS 1848, MD St. And. 1849
Evans, A., MRCS, LSA 1857
Evans, C. J., MRCS, LSA 1856
Evans, E. R., MRCS 1867
Evans, E. T., MRCS 1867
Evans, F. G., MRCS, LSA 1849
Evans, G., MRCS 1820
Evans, G. F., MD Cantab. 1839, FRCP 1859
Evans, H. N., MRCS 1860, MB Oxon. 1862
Evans, H. N., MD Glas. 1824, FRCS 1845
Evans, J., MRCS 1868
Evans, J. T., MRCS 1864, MD Aberd. 1865
Evans, L. E., MRCS 1867
Evans, N., MRCS 1859, MD St. And. 1859
Evans, O., MRCS 1827, MD Erlang. 1839
Evans, S. H., LSA, MRCS 1834
Evans, T., MRCS 1842, LSA 1844
Evans, T., MRCS 1837, LSA 1838
Evans, T., MRCS, LSA 1837
Evans, T. G., LSA 1823, MRCS 1825
Evans, T. M., MRCS, LSA 1861
Evans, W. L., MRCS 1862
Evers, C., MRCS 1861, MD St. And. 1862
Ewbank. F., MRCS, LSA 1867
Exell, E. MRCS 1866
Eyles, R. S., MSA 1812

Fairbank, T., MRCS 1864, MD Lond. 1865
Fairless, H. W., MRCS 1864
Faithorn, G., MRCS, LSA 1830
Falconer, R. W., MRCP 1860, MD Edin. 1859
Falwasser, F, MRCS 1858, FRCS 1866
Farncombe, W., MRCS 1852
Farr, J, in practice prior to 1815
Farrar, C., LFPS Glas. 1854, MD Heidl. 1854
Farre, A., FRS, MB 1833, MD Cantab. FRCP 1843
Farre, F. J., MD Cantab. 1837, FRCP 1858
Farrington, A. C., MRCS 1865
Farrington, W. H., MRCS 1859, MD St. And. 1862
Farwell, J., MRCS, LSA 1829
Favell, W. F., MRCS, LSA 1853
Fenn, E., MRCS, LSA 1861
Fenn, T. H., MRCS, LSA 1838
Fennell, T., MRCS 1865, LSA 1866
Fenton, H., MRCS, LSA 1844
Ferreday, T., LSA 1827, FRCS 1845
Ferris, G. T., MRCS 1837
Field, A. F., LSA 1868
Finch, J. E. M., MRCS 1865, LSA 1866
Firth, G. W. W., LSA 1835, FRCS 1845
Fish, J. C., MB Cantab. 1861
Fisher, F., MRCS 1856
Fitzgerald, C. E., MRCS 1856, MD St. And. 1862
Fletcher, M., LSA 1817, MRCS 1818
Folliott, J., MRCS 1863, LSA 1865
Foote, C. N, MRCS 1849, MD St. And. 1862
Foquett, H. R., MRCS, LSA 1852
Ford, B. B., MRCS 1860
Forder, J., MRCS 1818

Forshall, F. H., MRCS 1855, LRCP 1863
Forster, J. E., MRCS 1850
Fortescue, W., MRCS 1837, FRCS 1852
Foster, W. F., MRCS, LSA 1857
Fowke, F. G., LSA 1836
Fowler, O., MRCS 1835, LSA 1836
Fowler, O., MRCS 1863
Fowler, W., MRCS 1818
Fox, D., LSA 1821, FRCS 1843
Fox, G. F., MRCS 1843, LSA 1846
Fox, L. O., LSA 1833, MD St. And. 1859
France, W. H., MRCS 1860, LSA 1861
Francis, J., LSA 1834, MRCS 1835
Francis, M., MRCS 1846, LSA 1852
Franco, M. R. F., LSA 1832
Franey, E., MRCS, LSA 1858
Franklin, A. W., MRCS 1836, LSA 1837
Franklin, H., LRCP Edin. LSA. 1867
Freeman, J., LSA 1839, MD Erlang. 1841
Freeman, R. T., MRCS 1864, LRCP Edin. 1865
Freeman, S., LSA 1825, MRCS 1827
Freeman, T. A., LSA 1836, MRCS 1859
Freer, A., MRCS 1850, LSA 1852
Freer, R. L., MRCS 1839, LSA 1840
Freer, T., LSA 1822, MRCS 1828
Freer, W. C., LSA 1840, MRCS 1841
Fryer, T. W., MRCS 1854, LSA 1856
Fulcher, G. A., MRCS 1853, LSA 1853
Fullerton, J. C., LSA 1828
Furnell, M. C., MRCS 1850
Furley, E., LSA 1834, MD St. And. 1856
Furnivall, E. T., MRCS 1855

Gabb, A. W., MRCS 1843, LSA 1844
Gabb, D. H., MRCS 1845, LSA 1846
Gabb, F. E., MRCS, LSA 1862
Galton, J. C., MRCS 1866
Galton, R. C., MB Cantab. 1856, MRCP 1857
Gardiner, G. G., MRCS 1857, MD St. And. 1862
Gardner, J. T., LSA 1823, MRCS 1824
Gardner, S., MRCS 1861
Garlike, S. W. R., MRCS 1863
Garlike, T. W., MRCS 1839, FRCS 1858
Garlick, W., MRCS 1864
Garrett, W. J., MB Lond. 1867
Garrington, W. H., LSA 1826, MRCS 1845
Gaskell, R. A., MRCS, LSA 1849
Gatehouse, C., MRCS 1821, LSA 1819
Gatty, W. H., MRCS, LSA 1851
Gaved, A., MRCS 1846
Gay, J., MRCS 1811
Gay, J., MRCS 1841, LSA 1842
Gay, J., MRCS 1834, FRCS 1843
Gaye, C., MRCS 1819, LSA 1818
Gaye, H. S., MRCS, LSA 1850
Gaye, W., LSA 1818
Gayleard, J., LSA 1833, MRCS 1834
Gee, S. J., MD Loud. 1865
George, H., LSA 1850, MD St. And. 1862
George, H., LSA 1834
George, R. F., MRCS, LSA 1820
Geoghegan, R. T., MRCS 1863, LM Dublin
German, J., MRCS, LSA 1849
Gerrans, J. G., LSA 1831, MRCS 1832
Gibb, Sir G. Duncan, Bart. MD Montreal 1846, MRCP 1859
Gibbs, J. H., MRCS 1835, MD Aberdeen 1850
Gibbon, S., MB Cantab. 1851, MRCP 1852
Gibson, G., MRCS, LSA 1835
Gilbertson, J. B., MRCS 1849, MD Aber. 1859
Gilbertson, R., MRCS 1840, LSA 1841
Giles, W. F., MRCS 1858, LSA 1860
Gillard, F. J., MRCS 1837, LSA 1838
Gillott, E. D., LSA 1830, MRCS 1831
Gimson, W.G., MRCS 1860, MD St. And. 1862
Girdlestone, T. M., FRCS 1849
Girdlestone, W. T., MRCS 1856, LSA 1857
Gisborne, H. F., MRCS 1827
Glanville, J., MRCS, LM 1868
Glasier, G. W., MRCS 1859, LSA 1860
Glover, W. R., MRCS 1855, LSA 1861
Glynn, T. R., MRCS 1863
Goddard, L. M., LSA 1832, MRCS 1843
Goddard, R. C., MRCS 1864
Godfray, C. Le V., MRCS, LSA 1860
Godfrey, R., MRCS 1813
Godrich, F., LSA 1818, MRCS 1819
Godwin, A., MRCS 1855, MD St. And. 1862
Gooch, W. H., MRCS 1834, MD Edin. 1832
Goodacre, F. B., MB 1853, MD Cantab. 1860
Goodall, J., MRCS 1866, LSA 1867
Goodall, R., MRCS 1857, LSA 1859
Goodeve, H. H., MRCS 1828, FRCP 1860
Goodfellow, S. J., MD Lond. 1840, FRCP 1849
Goodsall, D. H., MRCS 1868
Goodwin, C., LSA 1834, MRCS 1845
Goodwin, J. W., MB 1849, MD Cantab. 1854
Goodwin, J. H., LSA 1836
Gore, R. T., LSA 1820, FRCS 1843
Gorham, R. V., LSA 1836, MRCS 1837
Gosse, J., MRCS 1868
Gostling, H. W., MRCS 1867
Goude, J. F., LSA 1837, MRCS 1838
Gover, R. M., MRCS 1856, LSA 1859
Gowar, F. R., MSA 1814
Graham, A. R., MC Cantab. 1863, MB 1864
Graham, T. H., MRCS 1841, LSA 1849
Graham, T. J., MRCS 1814, MD Glas. FRCP Edin. 1861
Grammer, S. R., MRCS 1850, LSA 1857
Grant, K., MD Edin. 1826, MRCS 1829
Gray, Charles, MRCS 1858
Gray, E. B., MRCS 1856, MD Oxon. 1861
Gray, T. S., MRCS 1855, MD St. And. 1857
Gray, W., MRCS 1855
Grayling, J., MRCS 1832, MD Aberdeen 1851
Greatrex, A. B. W., LSA 1852. MRCS 1855
Greatrex, A. B., LSA 1862, MD St. And. 1862
Greaves, W. T., MRCS 1831
Green, F. K., MRCS 1864, LSA 1866
Green, T., MD Edin. 1824, FRCS 1843
Green, J. C., MRCS, LSA 1834
Green, T., LSA 1822, MRCS 1823
Greenhill, J. R., MRCS 1860, FRCS 1864
Greeves, J., LSA 1833
Gregory, B., MRCS, LSA 1859
Grellett, C. J., MRCS, LSA 1864
Griffin, F. C. G., MRCS, MD Oxon. 1864

Griffin, R., LSA 1826, MRCS 1830
Griffith, S. C., MRCS 1864, MD St. And. 1847
Griffith, J. W., MRCS 1841, MD St. And. 1842
Griffith, T. T., LSA 1816, FRCS 1844
Griffiths, G. H., LSA 1849, MD St. And. 1862
Griffiths, W. H., MRCS 1861, LSA 1862
Grigg, N. B., MRCS 1860
Grime, H. A., MRCS 1840, LRCP Edin. 1860
Gross, J. E., MRCS 1857
Groves, Edward, MRCS, LRCP 1868
Gulliver, G., FRCS, 1843 FRS
Guppy, T. S., MRCS 1849, MD Aberd. 1859
Gutteridge, T., LSA 1826, MRCS 1827
Gwillin, W., LSA 1819, MD Erlang. 1831

Haffenden, T., MRCS 1807
Haig, J. B., MRCS 1842
Hall, E., MRCS, LSA 1846
Hall, E. F., MRCS 1858, MD St. And. 1860
Hall, F. R., MRCS 1852, LSA 1858
Hall, H. J., MRCS 1858
Hall, S., MRCS, LSA 1868
Hall, T. W., LSA 1839
Hall, W., LSA 1820
Hall, W., LSA 1840, FRCS 1862
Hall, W., LSA 1834, MRCS 1835
Hallowes, F. B., MRCS 1858, LSA 1860
Hallowes, P. B., LSA 1822, FRCS 1843
Hammond, A., LSA 1829, MRCS 1832
Hammond, C. C., LSA 1818, MRCS 1819
Hammond, E. C., MRCS 1860, LSA 1863
Hammond, F., MRCS 1860
Hammond, J., LSA 1832, FRCS 1843
Hanbury, C., jun., MRCS 1849, LSA 1850
Hands, B., LSA 1819, FRCS 1853
Hanne, J. J. A., MRCS 1868
Hanslip, T., LSA, 1853, MRCS 1859
Harbroe, E., LSA 1829, MRCS 1830
Harcourt, G., LSA 1821, FRCS 1841, MD St. And. 1846
Hardey, K., MRCS 1851
Harding, J. F., LSA 1835, FRCS 1852
Harding, W. W., MRCS 1855
Hardwicke, E. J., MRCS 1866
Hardwicke, H., MRCS 1840, LSA 1841
Harington, J. H., MRCS 1861, MB Oxon. 1862
Harper, T., LSA 1837, MRCS 1838
Harries, C. A., LSA 1829, MRCS 1830
Harris, A., MRCS 1862
Harris, B., MRCS 1860, LSA 1861
Harris, F., MB 1854, MD Cantab. 1859
Harris, J. S., MRCS 1868, LKQCP Irel. 1864
Harris, R. D., MRCS 1847, LSA 1856
Harris, S., MRCS 1854, LSA 1855
Harris, T., MRCS 1815
Harris, W., LSA 1831, FRCS 1858
Harris, W. J., MRCS 1859, LSA 1860
Harrison, C., MRCS 1860, MD St. And. 1861
Harrison, C. H. R., MRCS 1834, FRCS 1843
Harrison, E. P. D., MRCS 1842, LSA 1843
Harrison, R., MRCS, LSA 1859
Harsant, M., MRCS, LSA 1837
Harston, A. D., LSA 1839, FRCS 1859
Hartill, J., LSA 1826, MRCS 1856
Hartill, W. H., LSA 1860, MRCS 1861
Hartley, J., MRCS, LSA 1852
Harvey, O., MRCS 1860
Harvey, R. S., MRCS 1848, MD St. And. 1861
Harvey, W., MRCS, LSA 1834
Harvey, W., MRCS 1864
Haslam, G. H., MRCS, LSA 1861
Hatherly, N. C., MRCS 1845, MD Aberd. 1857
Havers, J., MRCS 1837, FRCS 1849
Haviland, H. J., MB 1849, MD Cantab. 1854
Haward, F. R., MRCS, LSA 1865
Haward, W., MRCS 1854
Hawett, T., MRCS, LSA 1822
Hawkes, J., MRCS 1853, MD St. And. 1861
Hawkins, C. J., LSA 1836, FRCS 1854
Hawthorn, F., LSA 1836, MRCS 1842
Hawthorn, H. O., LSA 1831, MRCS 1842
Haycock, G., MRCS 1862, LSA 1864
Haydon, N. J., MRCS 1837, MD St. And. 1860
Hayes, H., MRCS, LSA 1842
Hayman, H., MRCS 1834, FRCS 1858
Haynes, F. H., MRCS 1866
Hayward, J., MRCS, LSA 1845
Hazard, J., MRCS 1852, LSA 1857
Head, R. T., MRCS, LSA 1859
Head, T., MRCS 1822, MD St. And. 1845
Headley, W. B., MB & MC Cantab. 1864
Heaton, C. L., LSA 1837, MRCS 1838
Heaton, F. L., MRCS 1862, MB Oxon. 1863
Hedges, J. A., MRCS, LSA 1862
Heelas, M. L., MRCS 1866, LSA 1866
Hele, H. H., LSA 1825, MRCS 1826
Helm, G. F., MRCS 1859, FRCS 1863, MB Cantab.
Hemborough, J. W., LSA 1865, MRCS 1866
Henderson, H. G., MRCS 1863
Henderson, W., MD Aber. 1857, MRCS 1849
Henry, M., MRCS 1847, FRCS 1854
Hensley, P., MA, MB Cantab. 1867
Hepworth, G. A. H., MRCS, LSA 1847
Hester, J., MRCS 1853
Hester, J. T., LSA 1821, FRCS 1852
Hewer, E., MRCS 1866
Hewer, J. H , MRCS, LSA 1852
Hewlett, C., MRCS 1853
Hewlett, T., MRCS, LSA 1858
Hews, F., in practice prior to 1815
Hickens, G. L., MRCS 1854
Hickman, T. H., MRCS 1868
Hichens, J. S., MRCS, LSA 1851
Higgins, H., LM 1857, LRCP Edin. 1860
Hilder, H. H., LSA 1824, MRCS 1826
Hill, R. S., MRCS, LSA 1840
Hill, T. J., MRCS 1858, LSA 1859
Hill, W., MRCS, LSA 1842
Hillier, J. T., MRCS 1850, LM 1853
Hilton, C. S., LSA 1862, MD St. And. 1862
Hine, S. D., MRCS, LSA 1856
Hine, W. C., MRCS 1861, LSA 1862
Hingston, W., LSA 1824, MRCS 1825
Hingston, C. A , MB Lond. 1864
Hinton, J., MRCS 1848

Hitch, S., MRCS 1822, MRCP 1859
Hitchcock, C., MRCS 1834, LRCP Edin. 1860
Hitchcock, T., MRCS 1831, MRCP 1859
Hitchins, C. V., LSA 1854, MRCS 1855
Hoare, W., LSA 1826, MRCS 1827
Hoare, W., MRCS 1858
Hodges, F. S., LSA 1845, MRCS 1847
Hodgson, J., FRS, FRCS 1843
Hodgson, W., MRCS 1855, MD St. And. 1862
Hodson, C. F., MRCS 1837, LSA 1838
Holbrow, A., LSA 1818, FRCS 1862
Hole, J., LSA 1826, MRCS 1828
Hole, J. C., LSA 1822
Holden, G., MRCS 1856, MD St. And. 1859
Holden, L., MRCS 1838, FRCS 1844
Holland, Sir Henry, Bart., FRS, MD Edin. 1811; DCL Oxon, LLD Cantab.
Hollis, W. A., MB Cantab. 1867
Hollis, W. P., MRCS 1861, LM 1863
Holmes, A., MRCS, LRCSJ 1858
Holmes, C., LSA 1838, FRCS 1861
Holthouse, C., LSA 1832, FRCS 1848
Holttum, C., LSA 1840, FRCS 1855
Hooper, C., MRCS 1859
Hooper, J. D., LSA 1830
Hooper, J. T., MRCS 1845, LSA 1858
Hooper, L. G., MRCS 1852, LSA 1854
Hope, H., MRCS, LSA 1864
Hopgood, P. D., MRCS, LRCP 1867
Hopkins, H. C., MRCS 1867
Hore, H. A., MRCS, LSA 1844
Horne, T. B., MRCS 1845, LSA 1846
Horsfall, J., MRCS 1866
Hosking, R., MRCS, LSA 1852
Hoskins, E. J., MRCS 1858, MD St. And. 1860
Hoskins, H. R., LSA 1848, LRCP 1860
Houghton, J. H., MRCS, LSA 1838
Houseman, —, MRCS, LSA 1863
Howell, H. S., MRCS 1861, MD St. And. 1862
Hue, J. W., FRCP 1856, MD Cantab.
Hughes, D. W., MRCS 1861, LSA 1862
Hughes, J. B., MRCS 1866, LSA
Hughes, H. H., MRCS, LSA 1831
Hughes, H. R., MRCS 1839
Hughes, J., LSA 1840, FRCS 1858
Hughes, J. E., LSA 1861, MRCS 1862
Hughes, T. H., LSA 1858, MRCS 1858
Hughes, W. E., MRCS 1847, LSA 1857
Hughes, W. F., MRCS 1863
Hulme, E. C., MRCS 1844, FRCS 1849
Hulme, J. D., MRCS 1849, MD St. And. 1862
Humphry, C. H., MRCS, LSA 1862
Humphry, F. A., LSA 1853, FRCS 1856
Humphry, G. M., FRS, LSA 1842, MD Cantab. 1859
Hunt, E., LSA 1830
Hunt, H., MRCS 1840, FRCP
Hunt, T. O., MRCS 1856
Hunt, W. C., LSA 1844, MRCS 1845
Hunter, J. C., MRCS 1821, LRCP 1863
Hunter, R. H., MRCS 1861, LSA 1862
Hunter, T. D., MRCS 1825, LSA 1832
Hurlock, Rev. J., MD 1816
Husband, C., MRCS 1845, LSA 1846
Husband, H. A., MRCS 1866, MB Edin. 1867
Hussey, E. L., FRCS 1849, MRCP Edin. 1859
Hutchins, W., LSA 1825, MRCS 1826
Hutchinson, F., LSA 1828, FRCS 1852
Hutchinson, J., MRCS 1850, FRCS 1862
Hutchinson, J. R., MRCS 1845, LSA 1855
Hutchinson, T., MRCS 1849, LRCP Edin. 1860
Hutchinson, W.B., MRCS 1829, FRCS 1852

Iles, A., MD St. And. 1861
Iliffe, W., MRCS 1866, LSA 1867
Ilott, E., LSA 1848, FRCS 1863
Ilott, J. W., LSA 1836, MRCS 1837
Ingman, J., MRCS 1847
Ingram, C. P., MB Cantab. 1856, MD Cantab. 1860
Ingram, W. G. L., MRCS, LSA 1852
Irving, W., LSA 1830, FRCS 1852
Ireland, J., LSA 1853

Jebell, E. J., MRCS 1841
Jackman, T. S. H., MRCS 1848
Jackson, A., MRCS 1866
Jackson, H. W., MRCS 1855, LSA 1857
Jackson, J. H., MRCS 1856, MD St. And. 1860
Jackson, J. T., LSA 1841, MRCS 1842
Jackson, M., MRCS, LSA 1827
Jackson, M., MRCS, LSA 1866
Jackson, P., MRCS, LSA 1844
Jackson, R., MRCS 1832
Jackson, W., MRCS 1814, FRCS 1853
Jacob, E. L., MRCS, LSA 1855
Jacques, J. T., MRCS, LSA 1865
Jallard, R., MRCS 1854
James, E., MRCS, LSA 1853
James, E., MD Edin. 1835
James, E. M., MRCS 1860
James, H. H., LSA 1841, FRCS 1856
James, J., MRCS 1834, LSA 1862
James, J., MRCS 1862
James, J. D., MRCS, LSA 1857
James, J. H., MRCS 1811, FRCS 1843
James, T., MRCS, LSA 1836
James, T., MRCS, LSA 1846
Jarvis, N. H., MRCS 1867, LSA 1868
Jay, H. M., LRCP Edin., LM Edin., MRCS, LSA 1868
Jeaffreson, C., MRCS 1863, LSA 1867
Jeaffreson, H., MRCS 1860, MD Lond. 1865
Jeaffreson, J., MRCS 1862
Jeaffreson, J. B., MRCS, LSA 1858
Jeaffreson, W., MRCS 1820
Jefferys, W. E., LSA 1848, MRCS 1849
Jenneret, H., LSA 1824, MD Edin. 1825
Jenkins, H. J., MRCS, LSA 1829
Jenkyns, C., MRCS 1861, LSA 1862
Jepson, E. C., MRCS 1835, FRCS 1855
Jerrard, J. C., MRCS 1818, LSA 1819
Jeston, A. F. W., MRCS, LSA 1824
Johnson, E. R., MRCS 1867

Johnson, J. G., LSA 1820, FRCS 1843
Johnson, T. R. R., MRCS 1851, LSA 1854
Johnston, J., MRCS, LSA 1823
Johnstone, J., MD Cantab. 1832, FRCP 1834
Jones, A. B., MRCS 1848, LDS 1863
Jones, D., MRCS 1851, LRCP Edin. 1860
Jones, G. T., MRCS 1842, LRCP Edin. 1859
Jones, H., jun., MRCS 1853, LSA 1855
Jones, H. D., MRCS 1835, FRCS 1852
Jones, H. P., MRCS, LSA 1834
Jones, J., MRCS 1834, LSA 1838
Jones, J., MRCS, LSA 1862
Jones, M., MRCS 1855
Jones, M., MRCS 1856
Jones, M., MRCS 1861, LRCP 1861
Jones, O., MRCS 1862
Jones, R. O., MRCS, LSA 1861
Jones, R. A., MRCS 1864, LSA 1865
Jones, T., MRCS 1841
Jones, T., MRCS, LSA 1860
Jones, T. E., MRCS, LSA 1856
Jones, W., MRCS 1841, MD St. And. 1856
Jones, W., MRCS 1863
Jotham, G., LSA 1826, MRCS 1827
Jowers, F. W., MRCS 1854, LSA 1855
Julian, J. P., MRCS 1852

Karkeek, P. Q., MRCS, LSA 1865
Kay, J., LSA 1840, MRCS 1848
Keal, W., MRCS, LSA 1854
Kelly, J., MRCS, LSA 1833
Kemp, W. G., MRCS 1867, LRCP 1868
Kenys, A., LSA 1838, FRCS 1861
Kesteven, W. B., LSA 1837, FRCS 1854
Kiernan, F., FRS, MRCS 1825, FRCS 1843
Kiernander, W., MRCS 1864
King, E. P., MRCS 1858, LSA 1859
King, F., MRCS 1848
King, R., MRCS 1844
Kingdon, J. A., MRCS 1849, FRCS 1861
Kinsey, R. H., MRCS 1864, LSA 1866
Kipling, W., LSA 1827
Kirkman, J. M., LRCS Edin. 1833, MRCS 1835
Kirkman, J. M., MRCS 1867
Kitching, A., MRCS 1850, MD Aber. 1860
Kite, W. J., MRCS, LSA 1846
Knapp, F. W., MRCS 1864, LSA 1866
Knight G. T., LSA 1847, MRCS 1848
Knight, H. J., MRCS, LSA 1858
Knott, T. B., MRCS, LSA 1848
Kough, O'B., LSA 1860, MRCS 1861

La Farque, P. A., MRCS 1847, LSA 1854
Laity, R. J., MRCS 1837, LSA 1838
Lamb, B. W., MRCS, LSA 1866
Lancaster, W. J., MRCS, LSA 1860
Lanchester, H. T., MRCS 1861, MD Lond. 1864
Lanchester, T. W., LSA 1826, MD Erlang. 1839
Lang, W., MRCS, LSA 1832
Langdon, C. G., LSA 1866
Langdon, G. C, MRCS 1868
Langdon, H. W., MRCS 1854, LSA 1855
Langdon, T. C., LSA 1858, FRCS 1863
Langford, E. C., LSA 1855, MRCS 1856
Langshaw, J. P., LSA 1837, FRCS 1854
Langton, J., MRCS 1861, FRCS 1865
Langworthy, F. W., MRCS 1868
Langworthy, G. V., MRCS, LSA 1864
Langworthy, J. M. B., LSA 1852, MRCS 1854
Langworthy, R., MRCS, LSA 1836
Langworthy, S., MRCS 1851, LSA 1852
Langworthy, W. F., jun., MRCS, LSA 1861
Lansdowne, J. G., LSA 1827, MRCS 1828
Lascelles, R. M., MRCS, LSA 1836
Latham, C., MRCS, LSA 1839
Latham, J., MRCS 1823, LRCP Edin. 1860
Latham, P. M., MD Oxon. 1816, FRCP 1818
Latham, P. W., MRCP 1861, MD Cantab. 1864
Latham, R. G., MD Cantab. FRS 1850
Latten, R., LSA 1836, MRCS 1837
Lavery, T., LSA 1834, MRCS 1835
Lawrance, F., MRCS 1857, LSA 1859
Lawrence, G. R., MRCS 1862, LSA 1864
Lawrence, H. C., MRCS 1864, LRCP 1865
Lawrence, Sir J. Trevor, Bart. MRCS 1853
Lawrence, L. A., LSA 1839, MRCS 1843
Lawrence, R. M., MD Berl. MRCP 1849
Lawrence, W. F., MRCS 1841, LSA 1842
Lawton, J., LSA 1822, MRCS 1824
Lawton, R., LSA 1827
Laycock, W., MRCS 1837,
Leach, H., MRCS, LSA 1858
Leather, P. W., MRCS, LSA 1839
Ledgard, J. A., MRCS, LSA 1831
Ledsam, J. J., MRCS 1820, MD St. And. 1851
Lee, H. B., MRCS 1833
Lee, H., LSA 1830
Lee, J., LSA 1825
Lee, J., jun., MRCS 1858, LSA 1859
Lee, N. B. C., MRCS 1857
Lee, R., LSA 1825, MRCS 1843
Lee, T. W., MRCS, LSA 1868
Leech, E., MRCS, LSA 1822
Leech, H. P., MRCS 1848, LSA 1849
Leeds, T., MRCS, LSA 1861
Leggatt, R. S., MRCS 1841, LSA 1842
Leigh, P., LSA 1834, MRCS 1835
Lemon, H. M., LSA 1860, MD St. And. 1861
Leppington, H. M., LSA 1830, MRCS 1831
Lestourgeon, C., LSA 1831, FRCS 1843, MA Cantab.
Leverton, E. J., MRCS, LSA 1866
Leverton, H. S., MRCS 1851, LRCP Edin. 1860
Levy, J. L., MRCS 1866, LSA 1868
Lewis, D. W., MRCS 1861
Lewis, R. B., MB Lond. 1850, MRCS 1852
Lewis, W. T., MRCS 1853, LSA 1863
Ley, W., MRCS 1831
Lindop, J. C., MRCS 1862, LSA 1863
Lindop, W., LSA 1829, FRCS 1856
Lindgard, A. R., MRCS 1839
Lineker, E. H., MRCS 1858, LSA 1859
Linnecar, E. H., LSA 1824, MRCS 1843
Little, J. C., MRCS 1839, LSA 1840
Little, T. S., MRCS 1842, LSA 1843
Littleton, P. R., MRCS 1868

Livett, H. W., MRCS 1837, MD Pisa 1837, LRCP Edin. 1859
Llewellyn, E., MRCS 1859
Lloyd, D., MRCS, LSA 1838
Lloyd, D., MRCS, LSA 1859
Lloyd, J. A., MRCS 1829, MD Aberdeen 1854
Lobb, H., MRCS, LSA 1850
Lock, J. G., MA Cantab. MRCS 1867
Locock, Sir C. Bart. MD Edin. 1821, FRCP 1836
Lombe, T. R., MRCS 1843, MD Aberdeen 1856
Long, E., LSA 1823, MRCS 1824
Long, T., LSA 1833
Longhurst, A. E. T., MD St. And. 1862, MRCS 1863
Lorimer, J. A., MRCS 1868
Lovekin, L. F., LSA 1833, MRCS 1835
Loveless, W. K., LSA 1830, MRCS 1831
Lovell, F. H., MRCS 1865
Lowdell, C., MRCS 1847
Lowdell, LSA 1835, FRCS 1852
Low, A. J., MRCS 1864, LSA 1866
Lowe, LSA 1837, FRCS 1863
Lowne, B. T., MRCS, LSA 1844
Lowne, B. T., jun., MRCS 1861
Ludlow, E., MRCS 1862, MB London 1864
Ludlow, T., MD St. And. 1863
Luke, H., MRCS 1858
Lush, W. G. V., MRCS 1864, MB Lond. 1865

Maberly, G. S., MRCS 1866
Macaulay, F. E., MRCS 1861, LSA 1862
McCandlish, W., MRCS 1864
McCheane, W., MRCS 1843, LSA 1844
Macilwain, G., MRCS 1818, FRCS 1843
McKechnie, A., MD Glas. 1829, MRCP 1860
McKenzie, J., MRCS 1867
Mackenzie, J. T., MRCS 1858, MB Cantab. 1861
Mackinder, D., MRCS 1847, MD St. And. 1853
Mackintosh, H.R. D., MRCS 1863, MD Erlang. 1864
M'Clean, E. H., MRCS 1867
McGregor, A., MRCS 1867, LRCP Edin. 1868
McLeod, A. W., LSA 1827, MRCS 1828
M'Nab, D. R., LSA 1841, MRCS 1842
McNicoll, D. H., LSA 1836, MD Glasgow 1842
Madeley, G. S., MRCS 1867
Maling, E. H., LSA 1822, MRCS 1832
Mallet, S. E., MRCS 1861
Manby, A., LSA 1830, MRCS 1831
Manifold, W. H., MRCS 1849
Mann, R. M., MRCS, LSA 1845
Manning, J., LSA 1856, MRCS 1857
Mant, N., MRCS 1840, LRCP 1860
Marchant, R., MRCS 1815, LSA 1816
Margetson, P., MRCS, LSA 1835
Marrack, W., MRCS, LSA 1854
Marriott, J., MRCS 1850, LSA 1852
Marsh, A. J., LSA 1839, MRCS 1841
Marsh, F. H., MRCS 1861, FRCS 1866
Marsh, J., MRCS 1850, LSA 1856
Marshall, T. H., MRCS 1846, LSA 1849
Marson, J. T., LSA 1829, FRCS 1862
Martin, H. E., MRCS 1859
Martin, H. V., MRCS 1834, FRCS 1850
Martin, P., LSA 1863, MRCS 1864
Martin, Sir R., CB. FRS, FRCS 1843
Martin, R., MRCS 1813, FRCS 1844
Martin, R., MB 1851, FRCP 1859, MD 1859 Cantab.
Martyn, T. D., MRCS, LSA 1832
Maskelyne, H., LSA 1825, MRCS 1829
Mason, J. B., MRCS 1858, LRCP 1860
Mason, R., MRCS 1848, LSA 1856
Master, A., LSA 1837, MRCS 1838
Mathew, J. E., MRCS 1841, FRCS 1858
Mathias, J. E., MRCS 1838
Matterson, W., MRCS 1837, MD St. And. 1862
Matthews, J., LSA 1838, MD St. And. 1853
Maturin, H., MRCS 1864, LSA 1865
Maughan, J. B., MRCS 1840
Maxwell, E C., MRCS 1866, LSA 1867
May, A. S., LSA, MRCS 1864
May, E. C., LSA 1820, FRCS 1853
May, E. H., LSA 1854, FRCS 1856
May, J. H. S., MRCS, LSA 1859
May, J., LSA 1830, FRCS 1860
Mayne, R. F., MRCS 1854, MD Jena 1858
Mayo, C., MRCS 1861
Mayo, C., MRCS 1811, FRCS 1844
Mead, G. B., MRCS 1854, MD Giessen 1859, FRCP 1861
Meade, H., MRCS, LSA 1860
Meade, R. H., LSA 1835, FRCS 1845
Meldola, R., MRCS 1854, LSA 1860
Melhado, A. C. B., MRCS, LSA 1866
Meller, C. M., MRCS 1849, MD St. And. 1862
Menzies, J. J., MRCS 1859, LRCP Edin. 1860
Mercer, A. W., MRCS 1858, LSA 1859
Mercer, E., MRCS, LSA 1851
Merry, R. R., MRCS 1860, LRCP 1864
Metcalfe, E., MRCS 1841, FRCS 1859
Michell, G. A., MRCS, LSA 1835
Michell, S. V. P., LSA 1839, FRCS 1861
Mickle, D. K., LSA 1851, MRCS 1860
Middlemore, LSA 1825, FRCS 1843
Miles, C., MRCS, LSA 1848
Miles, H. C., MRCS 1854, LRCP Edin. 1859
Miles, J., LSA 1816, MD Erlang. 1837
Miller, C. M., MD St. And. 1862
Millington, LSA 1828
Milsome, J. R., MRCS 1862, MD St. And. 1862
Mitchell, S., LSA 1820, MRCS 1821
Mitchell, T. C., MRCS 1859, LSA 1860
Mitchinson, C. C., MRCS 1859, LSA 1860
Mitchinson, G., MRCS 1851, LRQCP Ireland 1859
Mitton, M. J., MRCS 1862
Moon, R., MRCS 1863
Monro, H., MD, MB Oxon. 1844, FRCP 1848
Montgomery, J. B., MD Glas. 1851, MRCS 1852
Moore, D. S., LSA 1841, MRCS 1842
Moore, E., MRCS, LSA 1851
Moore, E., LSA 1826, FRCS 1858

Moore, C. H., MRCS 1842, FRCS 1848
Moore, G. H., MRCS 1829, MD St. And. 1841
Moore, J., MRCS 1810
Moore, J., MRCS, LSA 1827
Moore, J., MRCS 1867
Moore, E., MD, LSA 1842, LRCP 1862
Moore, R. B., MRCS 1866, LSA, LRCP 1868
Moore, T., MRCS 1859, LRCP 1865, FRCS 1867
Moore, W., MRCS, LSA 1836
Moore, W. H., MRCS 1866
Moorhouse, J. W., MRCS 1852, LRCP Edin 1864
Morgan, W. F, MRCS 1828, FRCS 1848
Morris, D. E., MRCS 1868
Morris, E., MRCS 1854, LSA 1856
Morris, H., LSA 1831
Morris, R., MRCS 1857, LSA 1858
Morrish, T. F., MRCS, LSA 1861
Morse, A. C., MRCS 1844, LSA 1845
Mortimer, R., MRCS 1835, LRCP Edin. 1859
Moses, F. H., MRCS 1867
Moss, W. B., MRCS 1854
Mousley, G., LSA 1832, MRCS 1833
Moxon, J., MRCS 1857, LSA 1868
Mudge, B., MRCS 1863
Mudge, H., LSA 1828, MRCS 1829
Mullings, S. E., LSA 1854, LRCP E. 1860
Munday, C., MRCS 1847, LRCP Edin. 1859
Murrell, W., MRCS 1861
Muschamp, W. H, MRCS 1854
Myers, H. R., LM, LFPS 1859

Nash, W L., MRCS 1863
Nathan. H. F., MRCS 1860
Neatby, T., MRCS 1861, MD St And. 1861
Needham. F., MRCS 1858, MD St. And. 1862
Nelson, S., MRCS, LSA 1862
Ness, J., LSA 1826, FRCS 1860
New, H. S., MRCS 1835, LSA 1840
Newbold, E., MRCS 1835, LSA 1838
Newell, H. A., MRCS, LSA 1845
Newman, A., MRCS 1857, MB Oxon. 1859
Newman, G., MRCS 1835, LRCP Edin. 1859
Newman, H., MRCS 1841, LSA 1842
Newman, W, MRCS 1854, FRCS 1867, MD Lond. 1859
Newstead, C. H., MRCS 1867
Newton, E., LSA 1838, FRCS 1846
Newton, L., MRCS, LSA 1867
Nicholls, L., MRCS 1856, LSA 1859
Nicholson, J., LSA 1839, FRCS 1857
Nicholson, W. H., MRCS 1858
Noad, G. W, MRCS 1862, MD St. And. 1862
Noble, T., LSA 1858, MD Aberdeen 1859
Norris, H., MRCS 1813, FRCS 1844
Norris, T. G., MRCS, LSA 1832
Norris, W., LSA 1816, MD St. And. 1823
North, W. D. MRCS 1862
Norton, W. A., MRCS 1862, MD St. And. 1862
Nunn, J. R., MRCS, LSA 1859

Oates, J. P., LSA 1829, MRCS 1842
Odell, T., MRCS, LSA 1851
Odling, W., FRS, MB Lond. 1851, FRCP 1859
Oldham R., MRCS 1841, FRCS 1859
Oldman, J., MRCS, LSA 1858
Oliver, R., MRCS, LSA 1861
Oliver, W. H., MRCS, LSA 1836
Orford, W. C., MRCS 1847, LSA 1848
Orme, C., MRCS 1867
Ormerod, E. L., FRCP 1851, MD Cantab. 1851
Orton, G. H., MRCS 1863, FRCS 1867, MB Lond. 1867
Orton, W., LSA 1832, MRCS 1833
Osborn, H., MRCS 1851, MRCP 1859
Osmond, T., MRCS, LSA 1863
Overton, J., LSA 1821, MRCS 1822
Owen, A. P., MRCS, LSA 1835
Owen, E. R., LSA 1834, FRCS 1857
Owen, Professor Richard, FRS, MRCS 1826, FRCS 1843
Owen, R. J., MRCS, LSA 1861

Paget, G. E., MD Cantab. 1838, FRCP 1839
Paget, J., FRS, MRCS 1836, FRCS 1843
Paine, W., MRCS, LSA 1860
Palmer, C., LSA, MRCS 1866
Palmer, C, MRCS 1846, MD St. And. 1854
Palmer, E., MRCS 1839, MD St. And. 1845
Parke, J. T., MRCS 1848, LSA 1850
Parker, D., MRCS 1860
Parker, L., MRCS 1828, FRCS 1843
Parker, R., MRCS, LSA 1859
Parker, S., LSA 1840, MRCS 1841
Parks, J., MRCS 1834
Parry, P., in practice prior to 1815
Parry, R., LSA 1829, MD Aberd. 1848
Parsley, W. H., LSA 1831, MRCS 1862
Parsons, E. K., MRCS 1843, LSA 1844
Parsons, F. H., MD, CM Glas. 1861
Parsons, J. St. J. G., FRCS 1858, LRCP 1859
Parsons, W. A., LSA 1826, MRCS 1828
Partridge, J. H., MRCS 1840, LSA 1841
Partridge, R., MRCS 1827, FRCS 1843
Patten, C. A., MRCS 1861, LSA 1865
Pattinson, H. B., MRCS, LSA 1866
Paull, J., MRCS 1866, LSA 1867
Paxton, F. V., MB Oxon. 1864, MRCS 1866
Payne, G. S., LSA 1830, MRCS 1831
Payne, S, MRCS, LSA 1849
Pearce, F. D., LSA 1832, MRCS 1833
Pearce, F. D., jun., LSA 1862, MRCS 1863
Pearce, T., LSA 1825, MRCS 1826
Pearless, C. D., MRCS, LSA 1864
Pearse, W., LSA 1840, MRCS 1860
Pearse, W. D., MRCS, LSA 1836
Peatfield, T., MRCS 1863
Peel, R., MRCS 1861
Peirce, J. E., MRCS, LSA 1868
Peirce, R. K., MRCS 1844, LM 1855
Pemberton, O., MRCS, LSA 1847
Penfold, H., MRCS, LSA 1850
Pennington, A., MRCS, LSA 1861
Pennington, F., MRCS 1854
Pennington, T., LSA 1828, MRCS 1829
Pepler, W. B., MRCS, LSA 1853
Perkin, R. C., MRCS 1850, LSA 1851

Perrin, W. J., LSA 1825, MRCS 1856
Perrot, H., MRCS 1856
Perry, C. H., LSA 1835, MRCS 1843
Perry, J. G., MRCS 1824, FRCS 1843
Perry, M., MRCS 1866, LSA 1867
Pettifer, E. H., LSA 1861, MRCS 1862
Philbrick, S. A., MRCS, LSA 1830
Philbrick, T., LSA 1833, MD Giessen 1844, LRCP Edin. 1860
Phillips, E., MRCS 1839, MD Jena 1847
Phillips, G., FRCS 1855
Phillips, G. M., MRCS, LSA 1842
Phillips, J., LSA 1833, MRCS 1834
Phillips, J. W., LSA 1852, MRCS 1856
Phillips, W. E., MRCS 1867
Philpott, H. G., MRCS 1853, LSA 1854
Picken, S., MRCS 1855, LSA 1858
Pickett, J., MRCS, LSA 1858
Pickford, J., MRCS 1828, MD Aberdeen 1852
Pierce, J. E., MRCS 1868
Pierpoint, LSA 1855, MD St. And. 1862
Pilkington, T., MRCS, LSA 1863
Pilkington, W. H., MRCS 1845, LRCP 1861
Pinchard, B., MRCS 1843, MD Aberdeen 1859
Pinder, J. B., LSA 1855, MRCS 1856
Piper, C. C., MRCS 1848, LSA 1854
Pitt, E. G., LSA 1849, MD Aber. 1855
Place, J. L., MRCS, LSA 1866
Pocklington, E., MRCS 1860, LSA 1861
Pollard, J., MRCS, LSA 1854
Pollard, W., MRCS 1843, LSA 1844
Pollock, R. J., LSA 1826, FRCS 1852
Pooley, C., MRCS 1842, FRCS 1864
Porter, J. H., LSA 1827, M. D. Erlang. MRCP 1859
Potter, F. D., LSA 1832, MRCS 1835
Potter, T. H., LSA 1828, MRCS 1829
Pottle, E. G., MRCS 1867
Potts, W. J., MRCS 1861
Powdrell, J., MRCS, LSA 1864
Powell, A., MRCS 1832
Powell, F., MRCS 1853, MB Lond. 1855
Powell, H., LSA 1831, MRCS 1832
Powell, H., MRCS 1839, LSA 1840
Powell, J. J., LSA 1830, MRCS 1834
Powell, J. T., MRCS 1854, MD St. And. 1862
Powell, L., MRCS 1864
Power, F. D., MRCS 1866
Power, H., MRCS 1851, FRCS 1854
Power, W. H., MRCS, LSA 1864
Powne, B. L., LSA 1864, MRCS 1865
Powne, W., MRCS, LSA 1856
Pratt, G., MRCS 1845
Preston, W., MRCS 1855
Preston, W., MRCS 1866
Price, J. L., MRCS 1864, LSA 1865
Price, R. C., MRCS, LSA 1832
Price, W., MRCS, LSA 1821
Prichard, A., LSA 1840, MD Berlin 1841
Prichard, H. P., LSA 1834, MRCS 1835
Prichard, W., MRCS 1838, LSA 1839
Prideaux, A., MRCS 1834, LSA 1836
Pridham, J. W., MRCS 1860, LRCP 1862
Pridham, T. L., MRCS, LSA 1825
Prince, F. T., MRCS 1865, LSA 1866
Prince, T., LSA 1830, MRCS 1842
Pritchard, J. C., LSA 1826, MRCS 1843
Pritchard, J., MRCS, LSA 1849
Pritchett, G. M., MRCS 1839, LSA 1832
Pritchett, J. B., LSA, MRCS 1853
Proctor, A. G., LSA 1828, MRCS 1830
Proudlove, T. J., LSA 1834, LRCP 1859
Prowse, W., LSA 1848, MRCS 1849
Puddicombe, J. M., MRCS 1837, LSA 1838
Pugh, J. L. P., MRCS, LSA 1856
Pughe, H. R., MRCS, LSA 1857
Pullin, H. T. S., MRCS 1850, MD St. And. 1862
Pyne, R., LSA 1826, MRCS 1827

Quarrell, W., MRCS 1863
Quennell, J. C., MRCS, LSA 1859
Quick, J. R., MRCS, LSA 1836
Quick, J., MRCS, LSA 1866

Radcliffe, H. J., MRCS 1831, LSA 1830
Radley, W. H., MRCS, LSA 1857
Rae, Sir W. C. B., MRCS 1811, FRCS 1843
Randall, A. M., LSA 1821, MRCS 1822
Rainbird, H., MRCS 1856, LRCP Edin. 1860
Rainey, W. B., MRCS 1851, LRCP Edin. 1859
Randall, J., MB 1842, MD Lond. 1848
Randall, J. G., MRCS 1866
Randell, E. B., MRCS 1862
Raven, T. F., MRCS 1864
Ray, C., MRCS 1836, MD Pisa 1838
Raymond, L., MRCS 1852
Read, A., MRCS 1866
Read, C. G., LSA 1843, MRCS 1844
Reade, A. C., MRCS, LSA 1864
Ree, H. P., LSA 1839, FRCS 1858
Reece, R., LSA 1832, MRCS 1851
Reed, G., MRCS 1858, MD St. And. 1862
Reeve, J. F., MRCS 1812
Reeve, J. F., MRCS 1846, MD Aberd. 1857
Reid, J., MRCS 1843, LSA 1844
Reid, L. H., MRCS 1866
Rendall, C., LSA 1837
Rendell, R., MRCS. LSA 1832
Rendell, W. J., MRCS, LSA 1853
Rendle, J. D., MRCS 1846, MD St. And. 1862
Renshaw, B., MRCS, LSA 1868
Renshaw, E., MRCS 1866
Reynolds, T., MD Erlang. 1863
Reynolds, H. W., LSA 1822, MRCS 1843
Rhind, S., MRCS 1852, LRCP Edin. 1859
Rice, B., MRCS 1852, MB Lond. 1852
Richards, F. W., MB Lond. 1865, FRCS 1867
Richards, O., LSA 1834, MD Aberdeen FRCS 1848
Richardson, W. H., MRCS 1859, LSA 1860
Ridley, J. S., MRCS 1858, MD St. And. 1859
Rigden, G. C., MRCS, LSA 1839
Riley, H., LSA 1831, MRCS 1860
Riscoe, R. B., MRCS 1846
Robarts, H. P., LSA 1826, FRCS 1844
Roberts, J., MRCS 1834, MD Erlang. 1853
Roberts, R., LSA 1858
Roberts, T. E., MRCS 1864, MD Edin. 1864

Robertson, D. K., MRCS, LSA, LRCP 1868
Robinson, C., MRCS 1812, FRCS 1857
Robinson, C. R., MRCS 1846
Robinson, G., MRCS 1861, LM 1862
Robinson, H., MRCS 1861, LSA 1862
Robinson, R., MRCS 1866
Robinson, S. H., MRCS 1862
Rogers, A., FRCS 1853
Rogers, B., MRCS 1857, MD St. And. 1862
Rogers, G. O., MRCS, LSA 1826
Rogers, H. C., MRCS, LSA 1865
Rogers, H., MRCS 1848, LDS 1860
Rogers, H. J., MRCS 1852, LRCP Ed. 1862
Rogers, T. A., MRCS 1847, LDS 1860
Rogers, T. L., MRCS 1853, MD St. And. 1857
Rogerson, G., MRCS. LSA 1827
Rogerson, J., LSA 1827
Rolleston, G., FRS, MB 1854, MD Oxon. 1857, FRCP 1859
Rolls, T., MRCS 1861
Roper, C. H., MRCS, LSA 1850
Roper, G. S., MRCS, LSA 1836
Roscow, P, MRCS, LSA 1845
Roskrudge, T. H. A., LSA 1838, MRCS 1839
Rouch, J. R., MRCS 1868
Rouse, E., MRCS 1833, LSA 1836
Rowley, E. B., LSA 1825, MRCS 1826
Rowley, J., LSA 1834, MRCS 1837
Roworth, A. H., MRCS, LSA 1867
Royds, W. A. S, MRCS 1866
Ruddock. E H., MRCS 1868, MRCP Edin. 1865
Rudge, H., MD Erlang. 1814
Rummin, G., LSA 1859
Rumsey, C., LSA 1831, MRCS 1832
Rundle, H, MRCS, LSA 1866
Rusher, J. G., LSA 1841, MRCS 1842
Russell, G. I., MRCS 1820, MD Aberd. FRCS 1854
Russell, G. I., jun., MRCS, LSA 1860
Russell, J., MRCS 1857
Rust, H. R. G., MRCS 1865
Rye, A. B., LSA 1836, FRCS 1852
Rymer, J., MRCS 1815

Sadler, M. T. jun, MRCS 1855, MD Lond. 1861
Sadler, S. E., MRCS 1832, FRCS 1862
Salmon, F., MRCS 1818, LSA 1817
Samuel, R., MRCS 1868
Salter, J. R., MRCS, LSA 1852
Sandell, LSA 1830, MRCS 1831
Sanderson, H. J., MRCS 1842, MD St. And. 1853
Sandwith, H., MD St. And. 1834, FRCP 1859
Sandwith, H., MD St. And. 1850
Sankey, J. O., MRCS 1867
Sankey, W., MRCS 1844
Sankey, W. H. O., MD Lond. 1850, FRCP
Sargent, D. W., LSA 1836, MRCS 1837
Saul, W. W., MRCS, LSA 1867
Saunders, E. D., MRCS 1828, LSA 1829
Saunders, G., MRCS 1845
Saville, R., LSA 1836, MD Erlang. 1855
Savory, C. T., MRCS 1862, MD St. And. 1862
Savory, J. E., MRCS, LSA 1854
Savory, J. T., LSA 1823, MRCS 1824
Savory, W. S., FRS, MRCS 1847, FRCS 1852
Sawer, T., MRCS 1837
Sawkins, J., MRCS 1835
Scholfield, H. D., MRCS 1842, MD Oxon. 1847
Schollick, T. J., MRCS, LSA 1860
Scobell, Rev. G., MRCS 1853
Scobell, T. E., MRCS 1867
Scott, J., LSA 1833, FRCS 1854
Scott, R. J., MRCS, LSA 1844
Searle, G. C., LSA 1831
Sedgwick, H., MRCS 1866
Selwood, H. C., MRCS 1861, MD St. And. 1862
Selwood, J. H., LSA 1829, FRCS 1864
Senior, C., MRCS, LSA 1858
Sequeira, H. L, LSA 1852, MRCS 1859
Sewell, J. J., MRCS 1828, LSA 1847
Shackleford, G., LSA 1827
Sharp, D., MB Edin. 1866
Sharpe, H. J., MRCS 1862
Sharpe H. W., LSA 1850, FRCS 1862
Sharples, W., LSA 1834, MRCS 1835
Shaw, C. E. M., MRCS 1863, LSA 1864
Shaw, G. H., MRCS, LSA 1864
Shaw, H. E. F., MRCS 1844, LSA 1845
Sheehy, W. H., MRCS 1851, LRCP Edin. 1859
Shelton, H. D., MRCS 1867
Shephard, F, MRCS 1855, LRCP Edin. 1860
Sheppard, J. B., MRCS, LSA 1849
Sheppard, W., MRCS 1843, LSA 1844
Sherwin, W. L, MRCS 1859, LSA 1860
Shine, W. L., MRCS 1859
Shipton, J. N., MRCS 1855
Shurlock, M., LSA 1836, MRCS 1837
Sicard, A. B., MRCS, LSA 1831
Simons, T. R., MRCS, LSA 1833
Simons, J. A., MRCS 1847, MD St. And. 1862
Simpson, C. M., MRCS 1862
Simpson, J. H., MRCS, LSA 1866
Simpson, R. P., MRCS, LSA 1865
Simpson, S. H., MRCS, LSA 1864
Simpson, W., MRCS 1868
Simpson, W. I., LSA 1823, MRCS 1824
Sison, E. N., MRCS 1842, LSA 1844
Skeate, E., MRCS, LSA 1834
Skeel, D, LSA 1827, MRCS 1829
Skeel, T., LSA 1837, MRCS 1838
Skelton, J., sen., MD Mass. 1855, LRCP Edin. 1864
Skey, F. C., FRS, MRCS 1822, FRCS 1843
Sladden, J. A., MRCS, LSA 1834
Slaytor, J. C., LSA 1836
Sleeman, P. R., MRCS 1839, FRCS 1854
Sloman, S. G., LSA 1837, MRCS 1839
Slyman, M. D., MRCS 1855, LSA 1858
Slyman, W., LSA 1828, FRCS 1855
Smart, T. W. W., MRCS 1827, MRCP 1860
Smee, A., MRCS 1840, FRS, FRCS 1855
Smee, A. H., MRCS 1866
Smith, C. E., MRCS 1858, MD St. And. 1859
Smith, E. R., MRCS 1864, LSA 1865
Smith, E., MRCS 1834, LSA 1835
Smith, F. M., MRCS 1851, MD St. And. 1857

Smith, H., MRCS 1864
Smith, H. Spencer, MRCS 1837, FRCS 1843
Smith, J., LSA 1847, MRCS 1849
Smith, J. B., LRCS Edin. 1846, MRCS 1846
Smith, J. R., LSA 1824, MRCS 1859
Smith, J., LSA 1833, MD St. And. 1853
Smith, P., MD Aber. 1844, MRCP Lond. 1846
Smith, R. W., MRCS 1860, LRCP 1863
Smith, R., LSA 1828, MRCS 1829
Smith, T., MRCS 1854, FRCS 1858
Smith, W. A., MRCS, LSA 1856
Smith, W., MRCS, LSA 1834
Smith, W. J., MRCS, LSA 1857
Smith, W. A., MRCS 1856, MD St. And. 1857
Snook, J. W., MRCS, LSA 1864
Soame, C. B. H., MRCS, LSA 1854
Solly, S., MRCS 1828, FRCS 1843, FRS
Solomon, J. V., LSA 1838, FRCS 1854
Soper, J. H., MRCS 1855
Southey, R., MRCP 1860, MD Oxon. 1863
Southam, F., LSA 1841, MRCS 1842
Southam, G. T. M., MB Lond. 1863, MRCS 1862
Southam, T., LSA 1830, MRCS 1831
Southcomb, W. T., LSA 1824, MRCS 1825
Spackman, F. C., MRCS, LSA 1849
Spencer, E., in practice prior to 1815
Spencer, J. H., LSA 1835, MRCS 1836
Spicer, N. W., jun., MRCS 1853, LSA 1854
Spooner, E. O., LSA 1829, FRCS 1852
Spouncer, F. C., MRCS 1859, LSA 1860
Sprague, C. G, MRCS 1860
Spurway, C., MRCS 1863
Square, W., MRCS, LSA 1866
Square, W. J., MRCS, LSA 1835
Squire, A., LSA 1833, MRCS 1839
Stacpoole, G. C., MD Edin. 1845
Stafford, S. J. F., LSA 1858, MRCS 1851
Stallard, J. H., LSA 1842, MB Lond. 1858
Staning, R., MRCS 1837
Stanton, J., LSA 1832, MRCP 1859, MD Heidelb. 1842
Stanwell, W., MRCS, LSA 1855
Starke, A. G. H., MRCS 1859, MD St. And. 1861
Startin, J., LSA 1827, FRCS 1852
Starling, R. T., LSA 1824, FRCS 1856
Steel, C. D., MRCS 1836, FRCS 1859
Steel, J. S., MRCS 1843, MD Aberdeen 1854, FRCP Edin. 1855
Steele H. C. B., MRCS, LSA 1831
Stephenson, T. A., MRCS, LSA 1855
Stephenson, W., MRCS 1861, LSA 1862
Stevens, D. F., MRCS 1855, LSA 1856
Stevens, N. H., MRCS 1850
Stevenson, J. F., MD Edin. 1846, MRCS 1847
Stevenson, N., MRCS 1857, LDS 1862
Stewart, C., MRCS 1862
Stilwell, G., LSA 1827, MRCS 1828
Stilwell, H., MRCS 1857, MD Edin. 1857
Stilwell, J., LSA 1818, FRCS 1845
Stocker, G. S., MRCS 1831, LSA 1837
Stokoe, R., LSA 1828, MD St. And. 1862
Stone, D., LSA 1841, MRCS 1842
Stone, R. N., MRCS 1836, LRCP Edin. 1859
Stone, R. S., MRCS, LSA 1863
Stonehouse, C., LSA 1854, MRCS 1860
Stones, W. H., MRCS, LSA 1840
Storrs, R., MRCS 1856, LSA 1857
Stott, T. S., MRCS 1863, LSA 1864
Stowell, T., MRCS 1863, MD New York 1864
Stowers, N., MRCS 1838, LSA 1839
Stretton, S., MRCS 1854, LSA 1856
Stretton, W. H., MRCS 1851, LSA 1853
Strickland, E., MRCS, LSA 1860
Strutt, G. H., LSA 1860, MRCS 1861
Stubbs, H., LSA 1831, FRCS 1843
Sturkey, H. G., MRCS 1853, LRCP Edin. 1859
Stutter, F. A., MRCS 1853, MD St. And. 1860
Sugden, W., MRCS, LSA 1839
Summers, R. H., MRCS 1822
Sumner, W. A., MRCS, LSA 1842
Sutton, C. F., MRCS, LSA 1820
Sutton, F. J., LSA 1861, MRCS 1861
Sutton, H. G., MRCS, LSA 1860
Swales, E., LSA 1858, MRCS 1860
Swales, P., MRCS 1861, LSA 1862
Swan, W., BA Oxon, MRCS 1866
Swayne, S. H., MRCS 1843, LSA 1844
Swift, G., LSA 1824, LRCP Edin. 1863
Sylvester, G. M., MRCS, LSA 1839
Sympson, T., MRCS 1847, LRCP Edin. 1860

Taaffe, R. P. B., MRCS 1852, MB 1863, MD Lond. 1865
Tait, E. W, LSA 1851, MRCS 1852
Tanner, R. C., MRCS 1861
Taplin, B. D., MRCS 1854, LRCP Edin. 1860
Taplin, T., MRCS, LSA 1824
Tarleton, J. H., MRCS 1865, LSA 1866
Tate, R., in practice prior to 1815
Taunton, G., MRCS 1848, LSA 1852
Taunton, T., MRCS, LSA 1835
Tayler, C., LSA 1837, MRCS 1838
Tayler, G. C., MRCS 1866, MB Lond. 1866
Taylor, H., MRCS, LSA 1863
Taylor, J. E., MRCS 1845, LSA 1846
Taylor, J. S., LSA 1844, MD Aber. 1853
Taylor, T., MRCS 1842, LSA 1843
Taylor, T., MRCS 1818
Taylor, T., LSA 1821, FRCS 1861
Taylor, T., MRCS 1837
Taylor, W., MRCS, LSA 1830
Tattersall, W. J., MRCS, LSA 1866
Taynton, H., MRCS 1836, LSA 1839
Teare, T. M., jun., MD St. And., MRCS 1862
Tench, E. B., MRCS, LSA 1857
Terry, C., MRCS, LSA 1856
Terry, G., MRCS, LSA 1852
Terry, J., LSA 1825, MRCS 1826
Terry, J. J., LSA 1827
Thackwell, W., MRCS, LSA 1835
Theed, F., MRCS 1836, FRCS 1852
Thibou, J. W., MRCS 1864
Thomas, D. H., MRCS 1863, LSA 1865
Thomas, D. P., MRCS 1848, LSA 1851
Thomas, E., MRCS 1864
Thomas, J., MRCS 1836, LSA 1837

Thomas, J. B., MRCS 1864
Thomas, J., MRCS, LSA 1857
Thomas, J. H., MRCS 1850, MD Aberdeen 1860
Thomas, R., MRCS, LSA 1853
Thomas, R. H., MRCS, LSA 1853
Thomason, W. J., MRCS 1861, LSA 1860
Thompson, C. M., MRCS, LSA 1817
Thompson, C. R., MRCS 1850, LSA 1851
Thompson, C. T., MD St. And. 1855
Thompson, H., MRCS 1859, LSA 1860
Thompson, J. N., LSA 1824, MRCS 1825
Thompson, W. W., MRCS 1856, LSA 1857
Thomson, C. E., LSA 1828, FRCS 1863
Thorne, T. H., MRCS 1861
Thorne, R. T., MRCS 1863, MB Lond. 1866
Thornley, R. S., MRCS 1845, LSA 1846
Thurnall, W., MRCS, LSA 1838
Thursfield, R., LSA 1821, MRCS 1822
Thursfield, W., LSA 1824
Thurston, G. J., MRCS 1848, LRCP Edin. 1860
Tibbits, H., MRCS 1865, LRCP 1865
Tibbits, J., MRCS 1853, MD St. And. 1853
Tidboald, J. A., LSA 1827, MRCS 1828
Timmins, J. A. S., MRCS 1867
Tindale, A. McI., MRCS, LSA 1865
Tinsley, W. W., MRCS, LSA 1850
Tobin, G., MRCS 1867
Tomlinson, R. S., LSA 1827, MRCS 1828
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Toogood, T. B., MRCS, LSA 1833
Toogood, J., FRCS 1843, LRCP 1844
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Tracey, S. J., MRCS 1849, LRCP Edin. 1860
Tremearne, J., MRCS 1867
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Trollope, T. M. B., Cantab. 1854, MRCS 1856
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Twining, E., MRCS 1840, LSA 1841
Tyack, J., MRCS 1856
Tylecote, E., LSA 1826
Tylecote, E. T., MRCS 1855, MD Aber. 1860
Tyrer, R., MRCS 1843
Tyte, R. H., MRCS 1853

Upton, H. C., MRCS, LSA 1866
Upton, T. S., MRCS, LSA 1840

Vallack, A., MRCS, LSA 1836
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Valpy, W. H., LFPS Glas. 1853, LM Glas.
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Vardy, J., MRCS 1842
Vaudin, C., MRCS 1853, LRCP Edin. 1860
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Vernon, B. J., MRCS 1862, FRCS 1864
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Vickers, T., LSA 1831, MRCS 1833
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Vise, E., MRCS 1841, LSA 1842
Vise, E. B., in practice prior to 1815

Wadams, E., MRCS 1843, LSA 1845
Waghorn, F., MRCS 1862, MD St. And. 1862
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Walker, B., MRCS, LSA 1861
Walker, C., LSA 1846, MRCS 1848
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Walker, T., LSA 1834
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Watts, G., LSA 1852
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Wearing, A., MRCS, LSA 1864
Weatherly, F., MRCS, LSA 1841
Webb, G. F., MRCS, LSA 1865
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West, W. C., MRCS 1833, MRCP Edin. 1864, FRCS 1852
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Whidborne, G. F., LSA 1834, MD Heid. 1848
Whipple, C., MRCS 1863, LRCP 1864
Whitaker, E. F., MRCS, LSA 1824
Whitby, C. W., MRCS 1848, MB Aber. 1853
Whitcombe, E. B., LSA 1827, MRCS 1828
White, J. L., LSA 1836, FRCS 1845
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Whitney, W. U., MRCS, LSA 1835
Wicksteed, F. W., MRCS, LSA 1863
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Wilding, R., LSA 1838, MRCS 1839
Wilkins, E. P., MRCS 1847, LRCP Edin. 1859
Wilkins, H., MRCS 1835, LSA 1836
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Willett, A., MRCS 1859, FRCS 1862
Willett, C. V., MRCS, LSA 1861
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William, J., LSA 1860, MD St. And. 1861
Williams, A. W., MRCS 1843, MD St. And 1847
Williams, C., MRCS 1857
Williams, E., MRCS 1834, MD St. And. 1850
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Williams, E. H., MRCS, LSA 1861
Williams, E. E., MRCS, LSA 1860
Williams, H., MRCS 1815
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Williams, J., MRCS 1822, LSA 1823
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Williams, J., in practice prior to 1815
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Williams, O. W., MRCS 1820, MD Edin. 1840
Williams, P., MRCS, LSA 1856
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Williams, S. W. D., MRCS 1861, MD St. And. 1862
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Wilson, E., FRS, MRCS 1831, FRCS 1843
Wilson, F., MRCS 1858, MD Heid. 1858
Wilson, H. G., MRCS 1866
Wilson, J. A., MRCS 1859
Wilson, W. J., MRCS 1855, LRCP 1859
Wilton, F., MRCS 1855, LM 1856
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Wing, C., MRCS 1814
Wingate, R., MRCS 1858, LSA 1860
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Winstone, B., MRCS 1841, MD Aber. 1847
Winterbotham, L., MRCS, LSA 1856
Wingar, J., LSA 1826, MRCS 1827
Wise, R. S., MRCS 1840, MD St. And. 1842
Wiseman, W. W., MRCS, LSA 1833
Withers, F. O. B., MRCS 1859, LSA 1860
Witt, C., MRCS 1821, MRCP 1861
Witt, C., MRCS 1841
Witten, E. W., MRCS, LSA 1856
Wolferstan, S., LRCP 1864, MRCS 1864
Wolstenholme, J. H., MRCS 1851, LSA 1852
Wood, A. J., MRCS 1830, MD St. And. 1858
Wood, F., MRCS 1841, FRCS 1860
Wood, G. L., LSA 1818, MRCS 1819
Wood, J. Y., MRCS 1836, LSA 1837
Wood, T., MRCS, LSA 1827
Wood, T. O., MRCS 1868
Wood, T. C., MRCS 1840, MD Aber. 1846
Wood, W., MRCS, LSA 1857
Wood, W. J., MRCS 1858, LSA 1859
Woodhouse, J., LSA 1829, MD Aber. 1858
Woods, F. B., LSA 1830, MRCS 1856
Woods, F. H., MRCS 1859, LRCP Edin. 1860
Woodward, J. R., MRCS 1824
Wookey, J., MRCS 1858, LSA 1861
Woolhouse, H. H., MRCS 1849
Woollett, R. F., LSA 1838, MRCS 1839
Woolley, T. S., MRCS, LSA 1863
Worboys, F. T. S., MRCS, LSA 1862
Worger, T. H., MRCS, LSA 1864
World, R. R., LSA 1837, LRCP Edin. 1860
Wormald, T., MRCS 1824, FRCS 1843
Worship, J. L., MRCS, LSA 1847
Wotton, C., MRCS 1858, MD St. And. 1859
Wray, D. C., MRCS 1856, MD St. And. 1862
Wright, J., MRCS, LSA 1853

Wright, M., MRCS 1868, LSA 1865
Wright, T. G., LSA 1827, MRCS 1829
Wright, W. K., LSA 1852, FRCS 1852
Wrixon, J., MRCS 1842, LSA 1854
Wyer, O. F., MRCS 1858, LSA 1859
Wyman, G., LSA 1831, MRCS 1832
Wyman, J. S., LSA 1867
Wynne, J., MRCS, LSA 1834
Wynter, J. St. T., LSA 1830, MRCS 1858

Yarde, W., MRCS 1857, LSA 1858
Yarrow, G. E., MRCS, LSA 1863
Yates, J., MRCS, LSA 1846
Yates, W. H., MRCS 1851, LSA 1852
Yearsley, J., MRCS 1827, MD St. And. 1862
Young, R., MRCS 1818, MD Aberdeen 1851

*** In the above List the date of the earliest professional qualification is placed immediately after each name.

Gentlemen who desire their names to be added to this Register are requested to apply to the Honorary Secretary, who will be glad of any assistance in making it complete and correct.

EXAMINATIONS, 1868.

Senior Scholarship in Medicine, Surgery, and Midwifery—

1. A. F. Field
2. T. H. Hendley

Senior Scholarship in Anatomy, Physiology, and Botany—

1. H. Alder Smith
2. A. E. Cumberbatch

Junior Scholarship, 1867—

1. H. Alder Smith
2. A. E. Cumberbatch
3. C. J. Davis

Bentley Prize—
W. Deane Butcher

Kirkes Medal—
A. F. Field

Wix Prize—
A. F. Field

Hichens Prize—
A. F. Field

PRACTICAL ANATOMY, SEN.

Foster Prize—

A. E. Cumberbatch, H. Alder Smith
3. C. F. Gray
4. F. H. Bodman
C. P. Skrimshire, S. G. Sloman
7. W. G. Lowe
8. G. S. Payne
P. B. Stoney, H. Symons

PRACTICAL ANATOMY, JUN.

Treasurer's Prize—
W. J. Walsham

2. *Examiner's Prize—*
A. H. G. Doran
3. C. J. Newton
G. A. Phillips, W. R. Smith

ST. BARTHOLOMEW'S HOSPITAL

AND

MEDICAL COLLEGE.

The INTRODUCTORY ADDRESS will be given by Mr. THOMAS SMITH, on Thursday, October 1, at 2 P.M.

MEDICAL OFFICERS.

Consulting Physician—Dr. Burrows.
Physicians—Dr. Farre, Dr. Black, Dr. Edwards, Dr. Harris.
Consulting Surgeons—Mr. Skey, Mr. Wormald.
Surgeons—Mr. Paget, Mr. Coote, Mr. Holden, Mr. Savory.
Assistant-Physicians—Dr. Andrew, Dr. Southey, Dr. Church, Dr. Gee.
Assistant-Surgeons—Mr. Callender, Mr. T. Smith, Mr. Willett, Mr. Langton.
Physician-Accoucheur—Dr. Greenhalgh.

LECTURES.

WINTER SESSION, 1868-69.

Medicine—Dr. Black, Dr. Andrew.
Surgery—Mr. Paget, Mr. Coote.
Surgical Anatomy—Mr. Holden, Mr. Callender.
Physiology—Mr. Savory.
Chemistry—Dr. Odling, Dr. Matthiessen.
Clinical Medicine.—Dr. Farre, Dr. Black, Dr. Edwards, Dr. Harris.
Clinical Surgery.—Mr. Skey, Mr. Paget, Mr. Coote, Mr. Holden, Mr. Savory.

SUMMER SESSION, 1869.

Materia Medica—Dr. Farre.
Midwifery—Dr. Greenhalgh.
Botany—The Rev. G. Henslow.
Forensic Medicine—Dr. Southey.
Practical Chemistry—Dr. Odling, Dr. Matthiessen.
Comparative Anatomy—Dr. Church.
Dental Surgery—Mr. Coleman.

DEMONSTRATIONS.

Practical Anatomy—Mr. Baker, Mr. Langton.
Operative Surgery—Mr. Baker, Mr. Langton.
Morbid Anatomy—Dr. Church.
Diseases of the Skin—Dr. Andrew, Dr. Southey.
Diseases of the Ear—Mr. T. Smith.
Orthopædic Surgery—Mr. Willet.
Diseases of the Eye—Mr. Langton, Mr. Vernon.
Microscopic Anatomy—Mr. Vernon, Mr. Marsh.

Tutors—Dr. Duckworth, Mr. Baker, Mr. Shepard.
Medical Registrar—Dr. Southey.
Surgical Registrar—Mr. Willett.

COLLEGIATE ESTABLISHMENT.

Warden—Mr. BAKER.

Students can reside within the Hospital walls, subject to the College regulations.

Three Scholarships of £50 each, and four others varying in value from £20 to £30, are awarded annually.

Further information respecting Scholarships, Pupils' Appointments, and other details, may be obtained from Dr. Southey, Mr. Callender, or Mr. Morrant Baker, and at the Museum or Library.

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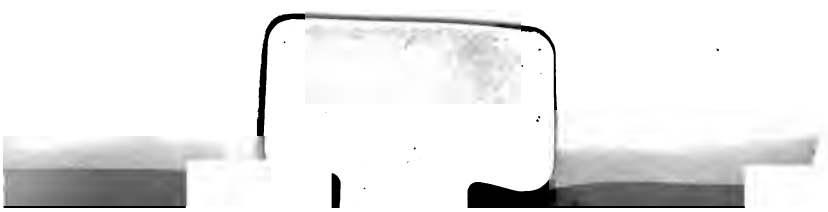
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